



Exam Preparatory Manual  
for Undergraduates

# Surgery

**Gunjan S Desai**  
**Ronak Patel**

*Foreword*  
**Shaji Thomas**

Exam Preparatory Manual  
for Undergraduates  
**Surgery**



# Exam Preparatory Manual for Undergraduates Surgery

A comprehensive review of surgery exam questions  
of various universities for undergraduates

**Gunjan S Desai**

MS General Surgery  
Delhi University

**Ronak Patel**

MD Radiodiagnosis  
Delhi University

*Co-authors*

**Suhani**

MS General Surgery  
Delhi University

**Tushit Mewada**

MCh Neurosurgery (Resident)  
GB Pant Hospital, New delhi



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## Jaypee Brothers Medical Publishers (P) Ltd

### Headquarters

Jaypee Brothers Medical Publishers (P) Ltd  
4838/24, Ansari Road, Daryaganj  
New Delhi 110 002, India  
Phone: +91-11-43574357  
Fax: +91-11-43574314  
Email: [jaypee@jaypeebrothers.com](mailto:jaypee@jaypeebrothers.com)

### Overseas Offices

J.P. Medical Ltd  
83, Victoria Street, London  
SW1H 0HW (UK)  
Phone: +44 20 3170 8910  
Fax: +44 (0)20 3008 6180  
Email: [info@jpmedpub.com](mailto:info@jpmedpub.com)

Jaypee-Highlights Medical Publishers Inc  
City of Knowledge, Bld. 237, Clayton  
Panama City, Panama  
Phone: +1 507-301-0496  
Fax: +1 507-301-0499  
Email: [cservice@jphmedical.com](mailto:cservice@jphmedical.com)

Jaypee Medical Inc  
The Bourse  
111 South Independence Mall East  
Suite 835, Philadelphia, PA 19106, USA  
Phone: +1 267-519-9789  
Email: [jpmed.us@gmail.com](mailto:jpmed.us@gmail.com)

Jaypee Brothers Medical Publishers (P) Ltd  
17/1-B Babar Road, Block-B, Shaymali  
Mohammadpur, Dhaka-1207  
Bangladesh  
Mobile: +08801912003485  
Email: [jaypeedhaka@gmail.com](mailto:jaypeedhaka@gmail.com)

Jaypee Brothers Medical Publishers (P) Ltd  
Bhotahity, Kathmandu  
Nepal  
Phone: +977-9741283608  
Email: [kathmandu@jaypeebrothers.com](mailto:kathmandu@jaypeebrothers.com)

Website: [www.jaypeebrothers.com](http://www.jaypeebrothers.com)  
Website: [www.jaypeedigital.com](http://www.jaypeedigital.com)

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## **Dedicated to**

*All the wonderful students  
who are going to be future doctors  
and who are the sole inspiration for writing this book*



# Foreword

---

In the MBBS training program, while there is a lot of stress on acquiring knowledge and skills, there is not enough emphasis on teaching students in handling the present format of theory question papers. Even 'good' students find a disconcerting 'disconnect' between the teaching-learning methods and the examinations. In teaching even the residents embarking on surgical careers, many of my colleagues and I, serving on medical school faculties across the country, were also all too aware of this disconnect.

This lovely book, expertly compiled by Dr Gunjan S Desai and his team, aims to bridge the gap between the standard teaching textbook and the theory questions that have to be answered by the student in his/her final summative examination.

Finally, this book is not meant to replace the standard textbooks of surgery, and, accordingly is not a comprehensive discussion of all surgical diseases. For an in-depth knowledge of surgical pathology, diagnosis and treatment, all students and especially the students embarking on surgical careers must first be thorough with a standard textbook of surgery before attempting to master this book.

I congratulate the authors on the successful outcome of their hard work and dedication, and hope the readers will find the book informative, useful and interesting to read.

**Shaji Thomas** MS

Director–Professor

Department of Surgery

Lady Hardinge Medical College

New Delhi, India



# Words of Appreciation from the Masters in this Field

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It gives me pleasure to learn that two of my students, Dr Gunjan S Desai and his team, have written a book *Exam Preparatory Manual for Undergraduates—Surgery* for MBBS students preparing for final professional surgery examination.

The book contains over 450 questions asked in various university examinations along with their answers in a simplified form.

I am hopeful that the book is helpful to the readers especially during revision phase of their preparation for final professional surgery examination.

I wish the authors and their readers all the best.

**Ajay Kumar** MS MCh FAIS FICS  
Director, Professor and Head  
Department of Surgery  
Lady Hardinge Medical College  
New Delhi, India

# Words of Appreciation from the Masters in this Field

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The book *Exam Preparatory Manual for Undergraduates—Surgery* is a combined effort made by a group of hard working, intelligent and competent residents from various fields of surgery.

The book covers around 450 questions and answers along with over 100 illustrations covering various sections of surgery.

I am confident and hopeful that the book turns out to be of great help for undergraduate students preparing for final professional examination.

I praise the efforts made by the authors and wish their endeavor a grand success.

**UK Shrivastava** MS FAIS DHA  
Former Professor of Surgery  
University College of Medical Sciences  
Dean and Head, Surgical Discipline, Faculty of Medical Sciences  
Chairman, Board of Research and Studies, University of Delhi  
Former Professor and Head, AIMST University, Government of Malaysia

# Preface

---

*“Surgery has always been and would always be a tough job  
whether it is performing a procedure or learning how to perform it.”*

When I entered the final year of MBBS, I faced this sentence for the first time. And now that I am a surgeon, I know how true it is. The reason is also slightly more obvious now. Just as there is no single standard technique to a single procedure, there is no single standard book to meet the university exam requirements.

My colleagues and I faced a great amount of difficulty in order to accumulate the material important for the final year MBBS university examination. Make the question lists of important questions, do peer reviews for the answers, search for authentic papers in net for answers and also the unauthentic material that we get hands-on during MBBS and finally when examinations came, we always thought that we were underprepared for the subject. The worst part of this exercise was not the reading part but the time that was spent in getting together the material to read.

The guidance in clinics is given for the cases and the practical part of the examination but not much is taught about the theory examination, which actually is necessary for the concept building. It is towards this end that I thought of writing this book as an aid to university examinations so as to make an effort to compile and accumulate all the material under one common heading. This book focuses more on the concepts that have been integral to getting the required knowledge of surgery and at the same time have a good compilation of all the university examination questions and their answers from authentic references.

So, I went in search of the appropriate people to help me in this herculean task and got together this splendid team of experts in this field and also experts at advising on the correct approach to master the concepts related to their field of interest in surgery. I thank all the team members for their hard work and contribution. Their insight in their sections in this book is definitely going to help everyone who uses this book.

The topics in this book are important for the university examinations and also important topics have been given a prolonged discussion to aid in the understanding of the topics. Also, the important conceptual points on clinical topics have been included for the student's benefit. I am sure that this book will be of great help to all the undergraduates, would save a lot of their time and effort and would make learning surgery fun.

**Gunjan S Desai**



# Acknowledgments

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We would like to thank the Almighty, our parents and family members, our teachers and all our seniors, colleagues and friends without whose support; this book would not have been possible.

We also thank Shri Jitendar P Vij (Group Chairman), Mr Ankit Vij (Group President) and Mr Tarun Duneja (Director–Publishing) of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, at the outset as without their support; this would have been a rather difficult task to accomplish. A special thanks also to Dr Sriram Bhat for contributing a few of the illustrations in this book.

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# How and When to Prepare for University Examination: An Overview

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Before we begin the journey into the field of surgery, we would like to congratulate all the readers to reach this stage in their medical career. The final year of MBBS is a tough year in all aspects of health—mental, physical and emotional and to remain healthy, you need proper planning and a good schedule to study and relax at appropriate times.

This book is a long sought solution to remaining “healthy” while preparing one big aspect of your final year—surgery: The subject with maximum mortality and morbidity in the MBBS field. Also, the subject is asked in different ways in examination and often is found tough to conquer. We are giving a schema to tackle this subject and this section is followed by all the important topics worth revising before examination and for entrance examination.

The best way to start the subject is to start as early as possible. Whenever you read any subject in MBBS, you need to decide two books for that subject—one with the details and one to get you through the examination. The book with details should be good enough to give you the basics and a grasp of subject with in-depth discussion of all the topics important for learning the subject. On the other hand, you also need a book to help in rapid revision and quick accumulation of various exam-oriented topics in the subject according to the university examination scheme to get you through the examination.

*Love and Bailey/Schwartz/Sabiston Textbook of Surgery* are considered standard descriptive books of surgery and are excellent to gain in-depth knowledge of the field but difficult to digest and revise at examination time. The list also extends to include *SRB's Manual of Surgery*, *Manipal Text of Surgery*, *Das Concise Text of Surgery* and many more. These books are descriptive and provide excellent insight regarding most of the topics of surgery even for postgraduates. This creates a rather difficult situation because both undergraduates and postgraduates are than reading the same basic material which does not seem right... Isn't it? Especially during examination, when you have to revise the book that you have first read, Harrison in Medicine and Bailey in Surgery, it is a tough task to complete, also with the thought in mind that you have other subjects to look after.

So, it is our advise to keep one textbook that suits you from the above options as a reference and this book since you start reading surgery and not just as a last resort book as this will help in rapid revision during the examination as well as necessary in-depth discussion of important topics as per undergraduate level.

## *How this book will help you in your preparation of surgery?*

- It will help you in gaining the requisite knowledge of all **important topics** of the field of surgery as per the last 10 years of university papers as well as **recent topics**, which are prospective questions in the years to come.
- This book also will provide you with the important and less frequently dealt topics of **Anesthesia and Radiology** that are not discussed in descriptive books and that need to be searched at examination times and create unnecessary waste of time and stress.



- Also, all the answers are from **latest editions of best surgical books** so as to help you in **clearing the surgery part of your MCQ preparation**.
- Because of the concise nature, it will help in **rapid revision** and will save time for other subjects.

So, begin early, read a descriptive book and a question-answer pattern book like ours to quickly revise and orient yourselves to all the important topics of the subject. This pattern of studies will give you the necessary confidence that you know the basics and also be equipped to clear the examinations without much duress. Apply this pattern to all the subjects to gain maximum benefit.

# SECTION

# 1

## General Surgery

- Wound Healing
- Fluid, Electrolyte and Acid-Base Imbalance
- Metabolism and Nutrition
- Blood Transfusion and DIC
- SIRS, Shock and MODS
- Basic of Surgical Technologies and Advanced Surgery
- Surgical Infections
- Trauma and Damage Control Surgery
- Perioperative Surgery
- Surgery of the Salivary Glands
- Miscellaneous General Surgery Topics of Importance
- Medicine in Surgery



## WOUND HEALING

**Q1. Write a note on mechanisms of wound healing.**

**What are the phases of wound healing? Discuss in brief.**

**Enumerate the phases and types of wound healing.**

**Ans.** Wound means the disruption of cellular and anatomic continuity of tissue.

**Goals of wound healing:**

- Regeneration (recovery of full structure and function without scarring)
- Seen only in bone and liver.

**Phases of wound healing**

1. Inflammation (1–3 days)	<ul style="list-style-type: none"> <li>• Hemostasis, chemotaxis, epithelial migration</li> <li>• Form fibrinous exudates/eschar</li> <li>• Chronic wounds become stalled in this phase</li> </ul>
2. Proliferation (3 days to 3 weeks)	<ul style="list-style-type: none"> <li>• Proliferation of endothelial cells, smooth muscle cells and fibroblasts</li> <li>• Granulation tissue</li> </ul>
3. Maturation (3 weeks onwards)	<ul style="list-style-type: none"> <li>• Contraction of wound edge</li> <li>• Scar and scar remodelling</li> </ul>

*Phase 1*

- Tissue injury causes activation of stromal cells and keratinocytes which on activation release macrophage chemoattractant protein (CCL 2), interleukin 8 (CXCL 8), CXCL 4,10 which are potent chemotactic for neutrophils
- Of these, CCL2, CXCL8 and 10 are elevated chronically in chronic wounds therefore keeping it in inflammatory phase.
- Interleukin 8 is low in fetal wounds which accounts for healing of wound with minimal inflammation and no scarring in fetal age
- C5a, leukotriene B4, C4 and D4 directly and via release of platelet aggregating factor from endothelium (thrombin mediates the stimulation of the endothelial cells to secrete PAF, interleukin1 and TNF) cause adhesion of the chemoattracted neutrophils. Interleukins and TNF are also released from monocytes and macrophages which also aid in this process
- Next after adhesion is migration of cells in extracellular matrix. Migration has phases of adhesion, spreading, contraction and retraction which are mediated by collagen, laminin and fibronectin in ECM with integrins on the cells
- Thus, by this mechanism, neutrophils finally reach the wound and mediate intracellular bacterial killing
- The role of these PMNs is only removal of contamination. It has no effect and no role in the process of wound healing.

One important event in this phase is of epithelialisation which has the phases of detachment migration, proliferation, differentiation and stratification and is mediated by EGF, KGF and TGF alfa.

*Phase 2*

- **Macrophages are the effector cells of this phase**
- Bacterial products, complement degradation products, C5a, thrombin, fibronectin, collagen, TGF beta and PDGF BB are all chemotactic for macrophages. Thus, the macrophages reach the wound site and mediate effects as follows:
  - Macrophages release interleukin 2, collagenase, elastase and in the presence of lipopolysaccharide, macrophage releases free radicals and mediate phagocytosis
  - It releases FGF, VEGF, TNF alpha and stimulates endothelial cell proliferation and angiogenesis
  - It also releases PDGF, EGF, IGF-1 and TGF beta and stimulates fibroplasias and collagen, elastin and glycosaminoglycan synthesis
  - It induces apoptosis of PMN
  - Releases IL1 alpha and therefore produces a febrile response.
- **Lymphocytes**
  - T lymphocytes induce fibroplasias by releasing TGF beta and TNF alpha
  - They also release interferon gamma and mediate effects such as downregulation of prostaglandin synthesis
  - Macrophage activation to release TNF alpha and IL1
  - Decrease collagen synthesis and keep macrophages in the wound
  - Interferon gamma is thus another important mediator in chronic non healing wound
  - B lymphocytes have no role in wound healing but are involved in downregulating wound healing once the wound closes.

**Two important events in proliferation phase are angiogenesis and fibroplasias****Angiogenesis**

- Activated endothelial cells degrade basement membrane of post capillary venules by release of plasmin and matrix metalloproteinases
- PDGF, TGF beta, FGF mediate migration of detached endothelial cells through these gaps and division resulting in tubule or lumen formation
- These tubules are then covered by deposition of basement membrane and capillary forms
- Important mediators include PECAM 1 which mediates endothelial cell-endothelial cell interaction and mediate cell-cell contact and beta1 integrin receptors which form tight junctions and stabilise the contacts developed by PECAM1 and lead to formation of capillaries, arterioles and venules
- Cell disruption and hypoxia are strong inducers of the angiogenic factors—VEGF (member of PDGF family) and PDGF are proangiogenic factors
- Timeline of angiogenesis
  - FGF2 provides the initial angiogenic stimulus at day 3
  - VEGF provides delayed and prolonged stimulus between days 4 to 7
  - TGF alpha and EGF causes cell proliferation
  - TNF alpha promotes formation of the capillary tube by increasing HIF 1 alpha which leads to increase in NO and VEGF during days 1 to 5.

*Phase 3*

- PDGF and FGF causes fibroblasts to become active which are called stimulated fibroblasts or myofibroblasts which then mediate this phase
- Actin appears at day 6 after wounding, persists at high levels till 15 days and then disappears by 4th week
- Matrix metalloproteinases are important in wound remodelling and contraction of all these, MMP 3 (stromelysin) is particularly important
- Epidermodermal interface in healed wound is devoid of rete pegs and therefore have increased fragility and avulsion after minnow trauma.

**Types of wound healing**

- **1st intention (Primary)** healing: Well approximated edges of incised wound heal by this way
- **2nd intention (Secondary)** healing: The wound is left open and allowed to heal on its own
- **3rd intention (Tertiary)** healing (Delayed primary closure): The wound is initially left open for dressing and then closed after few days.

**Q2. Write a note on pathophysiology and management of chronic nonhealing wound/ulcer in lower limb.****Enumerate the causes for the chronic nonhealing of a wound.**

**Ans.** Chronic wounds are wounds greater than 3 months.

These are the wounds which fail to proceed to functional and anatomic integrity over a period of 3 months.

**Pathology**

- Chronic wounds become stalled in inflammatory phase
- CCL2, CXCL 8 and 10 are elevated chronically in chronic wounds therefore keeping it in inflammatory phase
- Also increased levels of Interleukin 8, TNF alfa, Interleukin 1 and interferon gamma in these wounds along with increased MMP1,2,8,9 and decreased TIMP (inhibitor of matrix metalloproteinases) lead to decreased adhesion molecule expression, decreased cell migration, decreased growth factors and increased breakdown of products.
- This leads to increased inflammation and collagen degradation rather than collagen synthesis.

**Causes of nonhealing of wound**

- **Local infection is the most important factor to drive these mediators.** Organism count  $>10^5/g$  or single beta hemolytic streptococci are the most detrimental factors hindering wound healing. Other important causes for chronic nonhealing of wound include:
  - Hypoxia, anemia
  - Diabetes due to repeated trauma, tissue hypoxia due to vasculopathy, decreased VEGF/PDGF and HIF 1 alfa
  - Ionising radiation

- Aging
- Malnutrition—hypoalbuminemia (<2 g/dL), vitamin A/K deficiency, zinc deficiency
- Drugs—doxorubicin, nitrogen mustard, methotrexate, cyclophosphamide, bischloroethyl nitrosoureas, tamoxifen and steroids
- Ehlers-Danlos syndrome, osteogenesis imperfecta, epidermolysis bullosa, acrodermatitis enteropathica.

*Common nonhealing wounds include*

- Ischemic arterial ulcers and wounds—manages with revascularisation and wound care
- Venous stasis ulcers (post-thrombotic leg)—includes zinc oxide impregnated dressings with compression/4 layered dressing. Wound care is the most important aspect in venous stasis ulcer and is managed according to **Bisgaard's regime**. Surgery does not hasten or aid in wound healing (**ESCHAR trial**)
- Diabetic wounds management
- Pressure/decubitus ulcer management
- Foreign body in wound
- Neuropathic ulcers (sensory loss).

*Other causes include*

- Skin malignancy and marjolin's ulcer
- Vasculitis (SLE, PAN, rheumatoid arthritis)
- Osteomyelitis leading to chronic discharging sinus
- Infective causes, such as melioidoses, nontubercular mycobacteria (*M. fortuitum* and *M. ulcerans* cause bairnsdale/buruli ulcer, actinomycosis, MRSA)
- Immunosuppression (AIDS).

**General management measures include**

- History and physical examination
- Optimisation of general condition of the patient
- Ulcer edge biopsy/bone biopsy
- Chest X-ray/Mantoux testing
- Duplex imaging
- X-rays and MRI of the involved limb
- Treatment of underlying cause.

**Nonhealing wound local management**

- **Goal** is to convert wound from infected to clean to healing wound
- Dirty nonhealing wound should be evaluated under anesthesia, debrided as required, irrigated with free flowing saline and then hemostasis secured. Betadine is then applied on surrounding healthy skin and wound is dressed
- Tetanus prophylaxis and antibiotics should be administered as needed
- Routine dressings and maintenance of local hygiene is important to aid in wound healing

- If the wound is healing properly, then these measures are enough to promote wound healing
- If inspite of all the measures, the wound does not gets healed and all causes have been excluded, wound should be finally closed using biological dressings/excision and skin grafting.

**Q3. Enlist the factors affecting wound healing (discussed in que. Management of nonhealing wound and listed here).**

**Ans.**

- *Anatomic Site:* Fastest in areas of greatest blood supply (face and neck)
- *Weight:* Fat most vulnerable tissue to trauma due to poor blood supply
- *Age of wound:* Delayed healing, less wound strength
- *Oxygen:* Wound PO<sub>2</sub>, hyperbaric O<sub>2</sub>
- *Infection:* Local or systemic
- *Age of patient:* Fetal wounds heal the best > neonatal wound > adult wounds  
This is because of intrinsic fetal factors such as increased prolyl hydroxylase activity in fetal fibroblasts and extrinsic such as hyaluronan rich amniotic fluid
- *Medication:* Chemotherapy, steroids
- *Local factors:* Wound tension, foreign body, dead or devitalized tissue, excessive exudates.
- *Nutritional status:* Protein, zinc, vitamins, hypoalbuminemia (<2 g/dL)
- *Nature of wound:* Contusion, abrasion, laceration
- *Chronic disease:* Diabetes, cirrhosis, jaundice
- *Smoking:* Nicotine induced vasoconstriction, toxins, metalloproteinase stimulation
- *Radiation therapy:* Stasis and vessel occlusion
- *Hydration:* Moist wound heals faster
- *Growth factors:* Endogenous or exogenous growth factors stimulate wound healing. It is an area of active research—however, no magic bullet to date has been identified.
- *Congenital disorders:* Epidermolysis bullosa, Ehlers-Danlos syndrome, osteogenesis imperfecta
- *Malignancy, hypothyroidism.*

**Q4. Discuss the types of wound dressings.**

**Write a note on skin substitutes.**

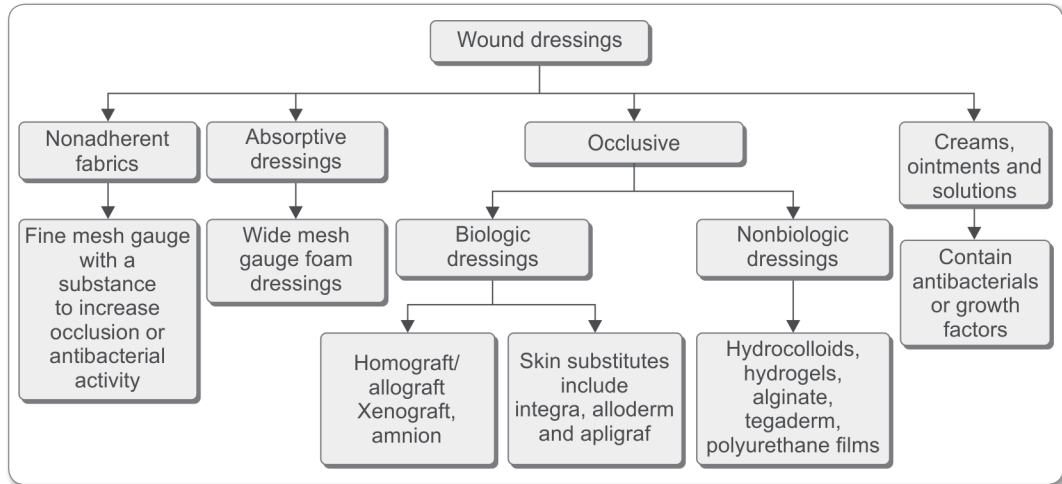
**Ans.**

**Aim:**

- To provide warm, moist and clean environment
- Barrier to bacteria and excess exudates should be removed
- Easy to use
- Cost effective
- Mechanical protection



## Classes



### *Absorptive dressings*

- Absorb excess exudates
- Disadvantages: Dressing becomes less effective when saturated and re-epithelialization is two times slower than occlusive dressings

### *Occlusive dressings*

- Mechanical protection
- Moisture retention
- Barrier to bacteria
- Provides mildly acidic pH and low oxygen tension which increases growth of fibroblasts and formation of granulation tissue.

### **Occlusive nonbiologic dressings**

- Hydrogels (polyethylene oxide)
- Alginate (sea weed)
- Hydrocolloids [gelatine, pectin, carboxymethyl cellulose (Duoderm)]
- Hydrocolloids are better than simple films because they have some absorptive properties.

### **Occlusive Biologic dressings**

1. Homograft/allograft—genetically identical to human beings
2. Xenograft—pig skin most common, others include bovine tendon collagen
3. Amnion—human placenta
4. **Skin substitutes:** Skin substitutes are biologic dressings that provide:
  - a. A structural support and
  - b. A scaffolding for regeneration

#### **a. Integra (Bilayered)**

- Silicone membrane on atmospheric side and bovine tendon collagen with chondroitin-6-sulphate on wound side

- Serves as a dermal template to induce fibroblast proliferation, granulation tissue formation and vascularization
- Once new collagen and dermal granulation is formed the silicone layer is removed and allograft is placed over it.

**b. Alloderm**

- Acellular dermal matrix from donated human skin
- Works same as integra by providing template for dermal formation. However, it would not provide a dermal matrix to support skin graft and therefore is inferior to integra.

**c. Apligraf**

- Living bilayered membrane to simulate human skin
- Neonatally derived dermal fibroblasts are cultured in collagen matrix for 6 days to form neodermis
- Human keratinocytes are then cultured on top of this neodermis
- This dressing contains cytokines and matrix but does not have melanocytes, macrophages, lymphocytes, Langerhan's cells or adnexa.

**Creams, ointments and special solutions**

Indications for their application include:

- Increased exudates
- Cellulitis
- Quantitative culture shows  $>10^5$  organisms/gram of tissue.

For example, include zinc oxide, silver nitrate, bacitracin, neosporin, acetic acid and growth factors.

**Q5. What is pressure sore? Discuss its management.**

**Ans.**

**Definition:** A sore area of skin that develops when the blood supply to it is cut off for more than two to three hours due to pressure on it and lack of movement.

Main causative factors thus include:

- Mechanical effects of prolonged pressure
- Shearing forces
- Prolonged immobilization

**Grading**

*NPUAP staging system for pressure ulcers*

<b>I</b>	Intact skin with nonblanchable redness of a localized area, usually over a bony prominence
<b>II</b>	Partial thickness loss of dermis appearing as a shallow, open ulcer with a red-pink wound bed and slough may also appear as an intact or open/ ruptured serum-filled blister
<b>III</b>	Full-thickness tissue loss, subcutaneous fat may be visible, but bone, tendon or muscles are not exposed
<b>IV</b>	Full-thickness tissue loss with exposed bone, tendon or muscle

*Contd...*

*Contd...*

<b>Unstageable</b>	Full-thickness tissue loss with the base of the ulcer covered by slough (yellow, tan, gray, green or brown) or eschar (tan, brown, or black) in the wound bed
<b>Deep tissue injury</b>	localized area of discolored, intact skin or blood-filled blister caused by damage to underlying soft tissue from pressure or shear

## Management

### *General measures*

- Air mattress/water bed
- Frequent change of posture
- Avoiding soiling by incontinence or drooling
- Nutrition of patient should be taken care of
- Immunonutrition
- Protection of all pressure points by glove balloons
- Release and treatment of contractures

Grade 1 and 2—general measures and moist dressings will take care of most of these pressure sores.

### **Options in treatment of grade 3 and 4 pressure ulcers**

As mentioned above, pressure sore grade 1 and 2 are managed with dressings and general supportive measures, whereas grade 3 and 4 usually require some form of reconstructive option once osteomyelitis or deep soft tissue infections are ruled out.

Direct closure and skin grafting don't serve much role in these grades of pressure sore.

### *Options thus include*

Sacral	Gluteus maximus myocutaneous flap Gluteal fasciocutaneous rotational or advancement flap
Ischial	Gluteus maximus myocutaneous flap Hamstring V-Y myocutaneous flap Posterior thigh flap based on inferior gluteal artery
Trochanteric	Tensor fascia lata advancement flap Rectus femoris flap Vastus lateralis myocutaneous flap
Unsalvageable	Hip disarticulation with tissue closure

## Intraoperative

- During surgery in patients with spinal cord injury, especially T5 or higher, care should be taken to avoid autonomic hyperreflexia while doing manipulation of pressure ulcer
- Flaps should be larger than wound and suture line should be far away from pressure points.

## Postoperative care

- Air mattress for 7 to 10 days
- Care while position changes

- Ischial flap—do not sit for 6 weeks
- Nutrition and muscle spasm control.

**Q6. Write a note on keloids.**

**Differentiate between keloid and hypertrophic scar.**

**Ans.**

Keloid	Hypertrophic scar
<ul style="list-style-type: none"> <li>• Genetic predilection is present</li> <li>• Predilection for darkly pigmented skin</li> <li>• No specific causative factors</li> </ul>	<ul style="list-style-type: none"> <li>• Not present</li> <li>• No such predilection</li> <li>• Foreign body in wound, wound infection, suturing under tension predispose</li> </ul>
<b>Pathology</b>	
<ul style="list-style-type: none"> <li>• Proliferation of immature fibroblasts and blood vessels with disordered collagen synthesis</li> <li>• Increased expression of TGF beta 1 and 2 with irreversible changes in extracellular matrix production</li> <li>• Center of keloid lesions contain a paucity of cells</li> <li>• Has increased vascularity</li> </ul>	<ul style="list-style-type: none"> <li>• Proliferation of mature fibroblasts and blood vessels and organized collagen synthesis</li> <li>• Hyperproliferation with normalisation on removing the causative factors</li> <li>• No such feature. Cells are uniformly distributed</li> <li>• No increase in vascularity</li> </ul>
<b>Clinical features</b>	
<ul style="list-style-type: none"> <li>• Females are more commonly affected</li> <li>• Occurs more commonly in a triangular region bordered by both shoulders and sternum</li> <li>• Grows beyond the borders of the original wound</li> <li>• Claw like extensions from the primary lesion are common</li> <li>• Itchy lesions</li> <li>• Can be painful and tender</li> </ul>	<ul style="list-style-type: none"> <li>• No sex predilection</li> <li>• No site predilection</li> <li>• Confined to boundary of wound</li> <li>• No extensions occur beyond the wound</li> <li>• Non-itchy lesions</li> <li>• No pain or tenderness</li> </ul>
<b>Natural history and prevention</b>	
<ul style="list-style-type: none"> <li>• Never regress spontaneously</li> <li>• Cannot be prevented</li> <li>• Difficult to treat</li> <li>• Recurrences are very common</li> </ul>	<ul style="list-style-type: none"> <li>• Regress spontaneously after 6–8 months</li> <li>• Can be prevented if causative factors are taken care of</li> <li>• Easy to treat</li> <li>• Does not recur</li> </ul>
<b>Common points</b>	
<ul style="list-style-type: none"> <li>• Both are hyperproliferation disorders with collagen abundance due to an imbalance between collagen synthesis and degradation</li> <li>• Both have stretched collagen fibers along the same plane as epidermis</li> <li>• Scars perpendicular to the direction of muscle fibers tend to be flatter and less chance of hypertrophic scars.</li> </ul>	

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Keloid	Hypertrophic scar
Prevention	
<p>Postsurgery scar management with silicone sheeting/triamcinolone acetonide at 6 weeks intervals/topical imiquimod cream or 5-Fluorouracil/pulsed dye lasers have the best outcome</p> <ul style="list-style-type: none"> <li>• First line treatment <ul style="list-style-type: none"> <li>– Triple therapy with surgery, steroid and silicone sheeting</li> <li>– Excision pulsed dye laser</li> <li>– Pressure therapy for 6 to 12 months</li> <li>– Intralesional steroid</li> <li>– Cryotherapy</li> <li>– Combination of either of these</li> </ul> </li> <li>• Second line treatment <ul style="list-style-type: none"> <li>– Radiation</li> <li>– Bleomycin therapy and tattooing</li> <li>– Interferon alfa-2, intralesional verapamil with surgery and silicone sheeting</li> <li>– Mederma gel (Onion extract gel with silicone)</li> </ul> </li> </ul>	

## FLUID, ELECTROLYTE AND ACID-BASE BALANCE

### Q7. Write a note on fluid balance in patients and surgery.

**Explain the normal water distribution in body.**

**Ans.**

- Total body water—60% of total body weight in males and 50% of total body weight in females (as females have more subcutaneous fat), most water is in skeletal muscles
- Of the total body water
  - 40% is intracellular
  - 15% is interstitial [includes 2% transcellular (water in CSF and joint spaces)]. The interstitial water has a rapidly equilibrating component and slowly equilibrating transcellular component
  - 5% intravascular (15% of this is arterial)
- Serum osmolarity =  $2 \text{ sodium} + \text{urea}/2.8 + \text{glucose}/18$  (Normal—280–310 mosm/kg)
- The tendency of solute exchange is determined by osmolarity
- The tendency of water exchange is determined by tonicity
- Tonicity is thus relative osmotic activity of two solutions, e.g. azotemia is a hyperosmotic condition but not a hypertonic condition
- Therefore osmolarity of the extracellular fluid is determined primarily by sodium, whereas the effective osmotic pressure between plasma and interstitium is determined by nondiffusible proteins and tonicity =  $2 \text{ sodium} + \text{glucose}/18$
- Osmolar gap = measured – calculated osmolarity which is due to ethanol, methanol, ethylene glycol and unidentified toxins
- Major intracellular cations include potassium, calcium and major intracellular anions include proteins and phosphates.
- Major extracellular cation include sodium and anions include chloride and bicarbonate.

**Fluid losses and gain in body**

Intake	2000–2600 mL/day
Urine	500–800 mL/day
Insensible loss	600 mL/day (75% from skin, 25% from lungs)
Stool	250 mL/day

**Important electrolyte values to remember**

In mEq/L	Sodium	Potassium	H+	chloride	Bicarbonate	Osmolarity
Sweat	50	5	50	50	0	
Gastric	50	0	90	100	0	
Bile	150	5	0	80	70	
Pancreatic	150	5	0	80	110	
Colon	120	30	0	80	30	
Ileostomy	100	10	0	50	50	
Diarrhea	50	50	0	20	40	
Ringer lactate	130	4	0	109	Lactate 28, calcium 3	273
Normal saline	154	0	0	154	0	308

**Q8. Enumerate the causes of hyponatremia.**

**Discuss the clinical features and management of a patient with hyponatremia.**

**Ans. Normal level**—135 to 145 mmol/L

**Causes**

Sodium and water loss	Euvolemic hyponatremia	Water and sodium excess (Water >> sodium)
<b>Renal</b> <ul style="list-style-type: none"> <li>• Diuretics</li> <li>• Mineralocorticoid deficiency</li> <li>• Osmotic diuresis (Glucose, mannitol, urea)</li> <li>• Renal tubular acidoses</li> </ul> <b>Extrarenal</b> <ul style="list-style-type: none"> <li>• Vomiting</li> <li>• Diarrhea</li> <li>• Burns</li> <li>• Pancreatitis</li> <li>• Rhabdomyolysis</li> </ul>	SIADH Hypothyroidism Psychogenic polydipsia Glucocorticoid deficiency	Nephrotic syndrome Congestive cardiac failure Cirrhosis Acute and chronic renal failure

**Clinical features**

- Headache, confusion
- Weakness, fatigue, muscle cramps
- Anorexia, nausea, vomiting, watery diarrhea, lacrimation, salivation
- Hypertension, bradycardia, oliguria

**Management**

- **Hypovolemic hyponatremia:** Isotonic saline administration

- **Euvolemic hyponatremia:** Water restriction
- **Hypervolemic hyponatremia:** Sodium and water restriction
- Free water excess should be corrected first and then correction of low sodium
- If neurological symptoms are present, they should be treated with 3% saline
- Otherwise, treat with 0.9% saline and the rate of treatment should not exceed 12 mEq/L/day as rapid correction can lead to **central pontine myelinolysis** now better known as osmotic demyelination syndrome which can have both pontine and/or extrapontine myelinolysis which causes seizures, weakness, akinetic movements and finally permanent brain damage
- **Formula for sodium deficit calculation**
- Sodium deficit = total body water (130 – measured)  
Volume—sodium deficit/154 (saline in liters)

**Q9. Enumerate the causes of hypernatremia.**

**Discuss the clinical features and management of a patient with hypernatremia.**

**Ans.**

**Causes**

Sodium and water loss	Euvolemic hyponatremia	Water and sodium excess (Sodium >> water)
<b>Renal</b> <ul style="list-style-type: none"> <li>• Loop diuretics</li> <li>• Osmotic diuresis (Glucose, mannitol, urea)</li> </ul> <b>Extrarenal</b> <ul style="list-style-type: none"> <li>• Diarrhea</li> <li>• Burns</li> <li>• Nasogastric aspirations</li> </ul>	Diabetes insipidus Insensible losses from skin and respiratory tract Psychogenic hypodipsia	Primary aldosteronism Cushing syndrome Hypertonic dialysis Bicarbonate infusion

**Clinical features**

- Restlessness, ataxia, lethargy, irritability, tonic spasm, delirium, coma
- Weakness, oliguria
- Dry sticky mucus membrane, red swollen tongue, decreased saliva and tears
- Tachycardia and hypotension

**Management**

- Initially, water deficit should be corrected first **then, hypernatremia correction is decided by following formula:**
  - Water deficit (liters) = total body water (sodium – 140)/140.
- Again, correction should not be done at a rate greater than 12 mEq/L/day.

Potassium balance is very important topic both for exams and for managing patients in clinics and therefore is explained in detail.

**Normal potassium homeostasis (Normal—3.5–4.5 mEq/L)**

- **Total body potassium** = 50 mEq/kg body weight. **Extracellular is 2% of this and only 0.4% is in plasma.** Rest is intracellular.

- Therefore, **1 mEq/L change in potassium in serum** is caused by either
  - An intracellular deficit of around 200–400 mEq **or**
  - Potassium excess of 100–200 mEq
- It is the value which is to be corrected to solve the patient's problem.

**Q10. Enumerate the causes of hypokalemia.**

**Discuss the clinical features and management of a patient with hypokalemia.**

**Ans.**

**Causes**

Renal causes	Extrarenal causes
<ul style="list-style-type: none"> <li>• Type I and II renal tubular acidoses</li> <li>• Acetazolamide</li> <li>• Diabetic ketoacidosis</li> <li>• Uretrosigmoidostomy</li> <li>• All causes of metabolic alkalosis</li> <li>• Hypomagnesemia</li> <li>• Bartter syndrome, Gitelman syndrome</li> </ul>	<ul style="list-style-type: none"> <li>• Diarrhoea</li> <li>• GI fistulas</li> <li>• Villous adenoma</li> <li>• Anorexia nervosa</li> <li>• Laxative abuse</li> <li>• Drugs</li> <li>• Hypokalemic periodic paralysis</li> <li>• Hyperaldosteronism</li> </ul>

**Spurious hypokalemia:** Extreme leucocytosis

**Redistributive hypokalemia:** Theophylline toxicity, barium toxicity, insulin therapy, etc.

**Drugs causing hypokalemia:** Aminoglycosides, amphotericin, cisplatin, foscarnet and ifosfamide

**Clinical features**

- Ileus, constipation
- Decreased reflexes, fatigue, weakness, paralysis
- Cardiac arrest and/or ECG changes as follows:

Flattened or inverted T waves, a U wave, ST depression and a wide PR interval. The prominent U wave is frequently superimposed upon the T wave and therefore produces the appearance of a prolonged QT interval.

- **Physiological variation:** Potassium decreases by 0.3 mEq/L for every 0.1 increase in pH above normal.

**Management**

- **First step is restoration of volume**
- **Symptomatic hypokalemia:** Give potassium 20 mEq IV over 1 hour under ECG guidance for maximum 4 doses. Rate of correction should not exceed 20 mEq/hour in any case and if at all it is required, it should always be done through **a peripheral venous line**. Maximum tolerated limit for potassium correction under ECG guidance is 100 mEq/hr.
- **If still not corrected:** Look for hypomagnesemia, as untreated hypomagnesemia can lead to resistant hypokalemia which will not be corrected till the magnesium level is corrected.
- **Asymptomatic patients:** Give oral syrup potassium chloride mixed with water, also add coconut water, bananas and other potassium rich sources.



**Q11. Enumerate the causes of hyperkalemia.****Discuss the clinical features and management of a patient with hyperkalemia.****Ans.****Causes**

<b>Spurious of hyperkalemia</b>	<ul style="list-style-type: none"> <li>• Tight tourniquet</li> <li>• Hemolysis</li> <li>• Leucocytosis/thrombocytosis</li> </ul>
<b>Redistribution of hyperkalemia</b>	<ul style="list-style-type: none"> <li>• Hyperglycemia</li> <li>• Succinylcholine, digitalis</li> <li>• Hyperkalemic periodic paralysis</li> </ul>
<b>High renin, low aldosterone</b>	<ul style="list-style-type: none"> <li>• Addison disease</li> <li>• Heparin and ACE inhibitors</li> </ul>
<b>Low rennin, low aldosterone</b>	<ul style="list-style-type: none"> <li>• Hyporeninemic hypoaldosteronism</li> <li>• Cyclosporine toxicity</li> </ul>
<b>End organ damage with high aldosterone</b>	Renal tubular damage due to: <ul style="list-style-type: none"> <li>• Autoimmune—SLE, amyloidoses</li> <li>• Hemolysis—sickle cell disease</li> <li>• Drugs—spironolactone, triamterene and amiloride</li> </ul>

**Drugs causing hyperkalemia:** Angiotensin receptor blockers, ACE inhibitors, digoxin, spironolactone, succinylcholine, heparin, cyclosporin, pentamidine, triamterene.

**Clinical features**

- Colic, diarrhea
- Nausea, vomiting
- Weakness, paralysis, respiratory failure
- Arrhythmias as follows:

Mild hyperkalemia (5.5–6.5 mEq/L)	<ul style="list-style-type: none"> <li>• Peaked T waves</li> <li>• Prolonged PR segment</li> </ul>
Moderate hyperkalemia (6.5–8 mEq/L)	<ul style="list-style-type: none"> <li>• Loss of P wave</li> <li>• Prolonged QRS</li> <li>• ST elevation</li> <li>• Ectopics</li> </ul>
Severe hyperkalemia (> 8 mEq/L)	<ul style="list-style-type: none"> <li>• Progressive widening of QRS complex</li> <li>• Sine wave appearance</li> <li>• Fascicular blocks/bundle branch blocks</li> <li>• Ventricular fibrillations</li> <li>• Asystole</li> </ul>

**Management**

- First step is restoration of volume.

*Therapies*

- **Calcium:** Used only for arrhythmia stabilization. It has no effect on potassium level. It should always be given with cardiac monitoring.

**2 solutions : Calcium gluconate 10%=0.5 mL/kg** slow IV injection or **calcium chloride 10%=0.1–0.2 mL/kg** slow IV injection.

- **Salbutamol: [K<sup>+</sup> sequestrant]**. Given by nebulisation or IV
- **Insulin/Glucose** to be given at the same time (**K<sup>+</sup> sequestrant**)
- **Bicarbonate:** In metabolic acidosis only
- **Dialysis (K<sup>+</sup> excretion)**
- **Kayexalate (K<sup>+</sup> excretion)**

### Severe hyperkalemia

Any patient with K<sup>+</sup> >7.0 mEq/L or at risk of increasing and/or patient symptomatic and/or ECG disturbance:

*Manage with the following measures:*

- Calcium IV if ECG changes, salbutamol nebulization, insulin/glucose IV, bicarbonate IV if metabolic acidosis
- Dialysis
- Kayexalate (Polystyrene sulfonate) PR (if dialysis unavailable)

### Moderate hyperkalemia

Any patient with K 6 to 7 mEq/L, asymptomatic with normal ECG

*Manage with following measures:*

- Salbutamol nebulization, insulin/glucose IV, kayexalate (polystyrene sulfonate) PR or oral, bicarbonate IV if metabolic acidosis

### Mild Hyperkalemia

Any patient with K >5.5, asymptomatic with normal ECG

*Management*

- Stop K supplements
- Salbutamol nebulization, bicarbonate IV if metabolic acidosis.

## Q12. Enumerate the parameters useful to decide acid-base disorders and give their normal values.

**Answer**

pH	7.35–7.45
PaO <sub>2</sub>	74–82 mm Hg
PaCO <sub>2</sub>	35–45 mm Hg
SaO <sub>2</sub>	>92%
CaO <sub>2</sub>	16–20 vol %
HCO <sub>3</sub> <sup>-</sup>	22–26 mEq/L

### Important buffering systems in body

- Intracellular—proteins and phosphates
- Extracellular—bicarbonate-carbonic acid system

## Q13. Discuss the steps of diagnoses of an acid-base disorder in a patient.

**Explain the mechanism of identification of an acid-base disorder and show its compensation mechanism.**

**Ans.** Acid base imbalances are diagnosed as follows:

**1st step:** Identify the primary acid-base disorder (Mnemonic—ROME – **R**espiratory has **O**pposite change between pH and  $pCO_2$  whereas **M**etabolic has **E**qual direction change between pH and bicarbonate)

ABG	pH	$PaCO_2$	$HCO_3$	Compensation
Metabolic acidoses	Decrease	N	Decrease	Decrease in $PaCO_2$
Metabolic alkalosis	Increase	N	Increase	Increase in $PaCO_2$
Respiratory acidoses	Decrease	Increase	N	Increase in $HCO_3$
Respiratory alkalosis	Increase	Decrease	N	Decrease in $HCO_3$

**2nd step:** Identify compensation as shown in the chart above

**3rd step:** See if gaps are present in case of metabolic acidoses

- Anion gap

Unmeasured cations (UC) (Mn: PCM)	Unmeasured anions (UA) (Mn: SOAP)
Potassium, calcium and magnesium	Sulphate, organic acids, albumin, phosphate
<b>Total</b> – 11 mEq/L	<b>Total</b> – 23 mEq/L

Anion gap = UA – UC = 12 mEq/L = sodium – (chloride + bicarbonate)

Adjusted anion gap =  $2.5 (4.5 - \text{albumin}) + \text{observed anion gap}$ .

High anion gap acidoses	Normal anion gap acidoses
Ethanol/ methanol/propylene glycol poisoning Salicylates Isoniazid toxicity Ketoacidoses (Diabetes/starvation/alcohol) Lactic acidoses Uremia	Proximal (Type 2), distal (Type 1) and Type 4 Renal tubular acidoses Early renal failure Exogenous ammonium chloride or HCl Administration Diarrhea Carbonic anhydrase inhibitor therapy

- **Gap-gap in acidoses: It is calculated only when anion gap is present**

Gap-gap = anion gap excess/bicarbonate deficit =  $(\text{measured anion gap} - 12) / (24 - \text{measured bicarbonate})$

- If gap-gap < 1, it indicates co-existence of normal anion gap acidoses with high anion gap acidoses
- If gap-gap > 1, it indicates co-existence of metabolic alkalosis with high anion gap acidoses.

**Clinical features of acid-base imbalance are as shown in the table on the next page:**

**Q14. Write a note on metabolic acidoses (above matter + following matter will make the SN).**

**Ans.**

- First step is restoration of volume
- Management mainly aims at correction of the underlying cause
- Sodium bicarbonate is given only when pH < 7.1/sodium bicarbonate < 15 mEq/L
- **Base deficit = 0.6 body weight (i.e. TBW) (15 – sodium bicarbonate) in mEq/L**
- Half of this value is administered bolus and half added in the infusion form
- However, sodium bicarbonate has dubious role in management of metabolic acidoses less severe than this and especially in patients with lactic acidoses.

Symptoms of acidosis	Symptoms of alkalosis
Headache, sleepiness, coma, dyspnea, arrhythmias, nausea, vomiting, diarrhea, seizures, weakness	Nausea, vomiting, muscle twitches, tremors, numbness, confusion, coma, tingling in hands and feet.

**Q15. Explain: Metabolic alkaloses.****Ans.****Causes**

Chloride responsive	Chloride unresponsive
<ul style="list-style-type: none"> <li>Respond to chloride administration</li> <li>Has low chloride in plasma and urine</li> </ul>	<ul style="list-style-type: none"> <li>Does not respond to chloride administration</li> <li>Does not have low chloride in plasma and urine</li> </ul>
<ul style="list-style-type: none"> <li>Post-hypercapnea</li> <li>Diuretic therapy</li> <li>Villous adenoma</li> <li>Gastric aspirations</li> </ul>	<ul style="list-style-type: none"> <li>Laxative abuse</li> <li>Bartter syndrome</li> <li>Primary aldosteronism</li> <li>Cushing syndrome</li> <li>Malignant or accelerated hypertension</li> </ul>

**Management**

- First step in management of any fluid electrolyte or acid-base disorder is the correction of fluid abnormality, i.e. hypovolemia
- Final management depends on correction of cause
- Chloride deficit = 0.3 body weight (100 – chloride level)**  
Volume to replace = chloride deficit/154 in liters of isotonic saline
- H<sup>+</sup> deficit = 0.5 body weight (actual – desired bicarbonate)**  
Volume to replace = H<sup>+</sup> deficit/100 in 0.1N HCl.

**Q16. Write a note on plasma expanders.****Ans.****Rationale**

- Crystalloids redistribute after intravenous administration in a ratio of 1:3 that is for 1 part that remains in the intravascular compartment, 3 parts go into the interstitium. For example, if 1 liter crystalloid is administered, 750 mL goes into interstitial space and 250 mL remain in intravascular space
- Colloids on the other hand distribute 3:1, that is opposite of crystalloids and thus are called plasma expanders. These are large molecules with poor diffusibility which create an osmotic pressure to increase fluid in intravascular space
- Therefore to counteract blood loss, volume to be transfused
- Crystalloid—3:1
- Colloid—1:1

**Plasma expanders include**

- 5% albumin
- 25% albumin (latest)
- 6% dextran
- 6% hetastarch

**Conflicts**

- No convincing evidence that colloids are better than crystalloids.
- No convincing evidence that one colloid is better than other colloid.

**Adverse events**

- Decreases immunoglobulin response
- Decreases albumin production
- Decreases ionised calcium level
- Decreases response to tetanus toxoid
- Increases ECF volume deficit
- Dextran interferes with cross matching and it causes coagulopathy
- Hetastarch decreases vWF and factor 8c levels and causes coagulopathy
- Hetastarch also causes macroamylesema.

25% albumin has been found to have some advantages over other colloids in new studies

- It has proven anti-inflammatory effect, volume requirement is 5 times less and it has no coagulopathic side effects
- However, 25% albumin causes only fluid shift in body. Therefore, it should not be used in cases of acute volume loss unless some volume is restored

Hetastarch: It is a starch polymer (6%) in isotonic saline

- High molecular weight—4.5 lacs
- Medium molecular weight—2 lacs
- Low molecular weight—70,000 daltons
- Hextend—6% hetastarch solution with a buffered multi-electrolyte solution

Dextran 70 has a longer duration of action than dextran 40

- None of these fluids have definite preferential advantages over others
- All of them have minimal risk of allergic/anaphylactic reactions.

## METABOLISM AND NUTRITION

**Q17. Explain the endocrine and metabolic response to stress/surgery/injury.**

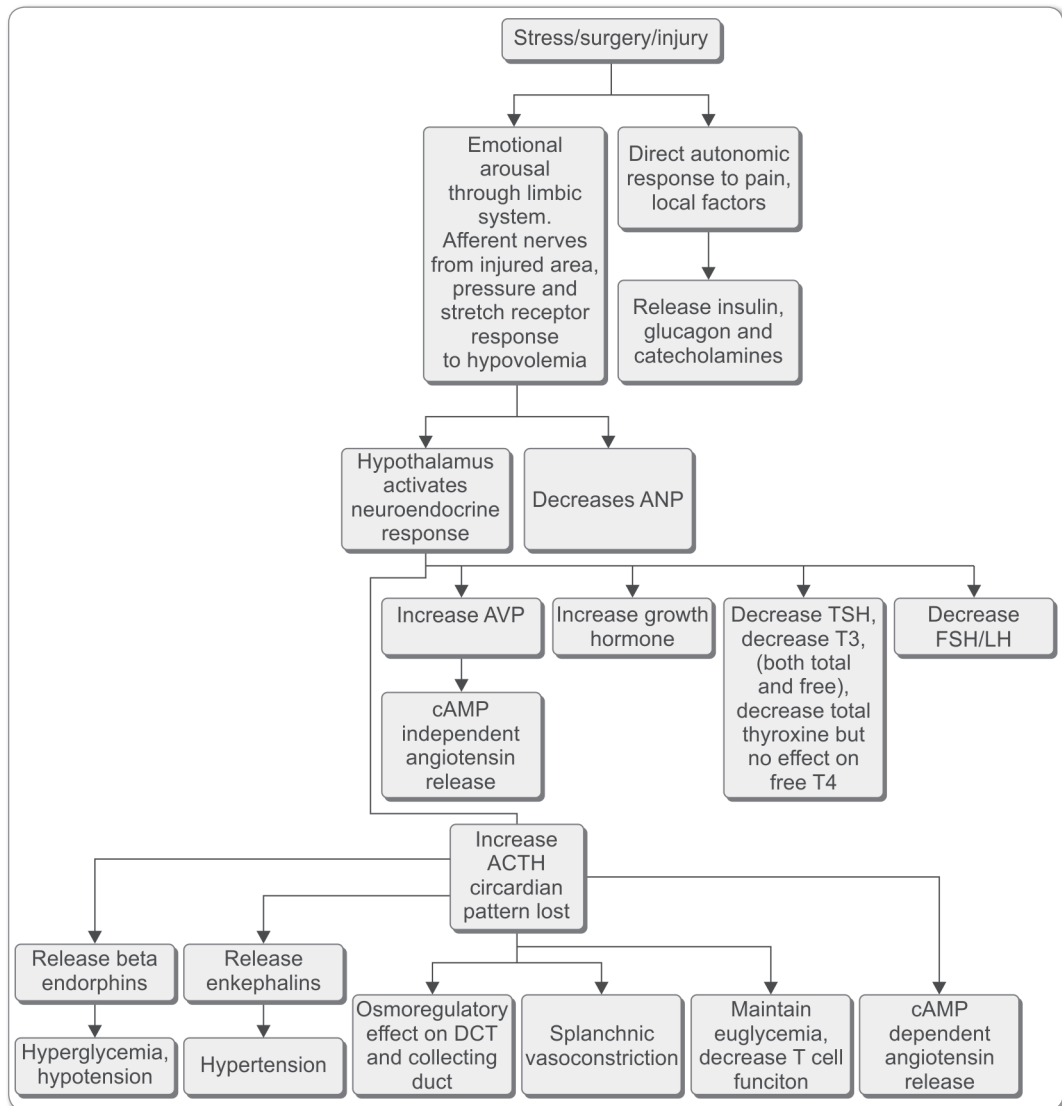
**Ans.**

**Effect of stress on pancreas is as follows:**

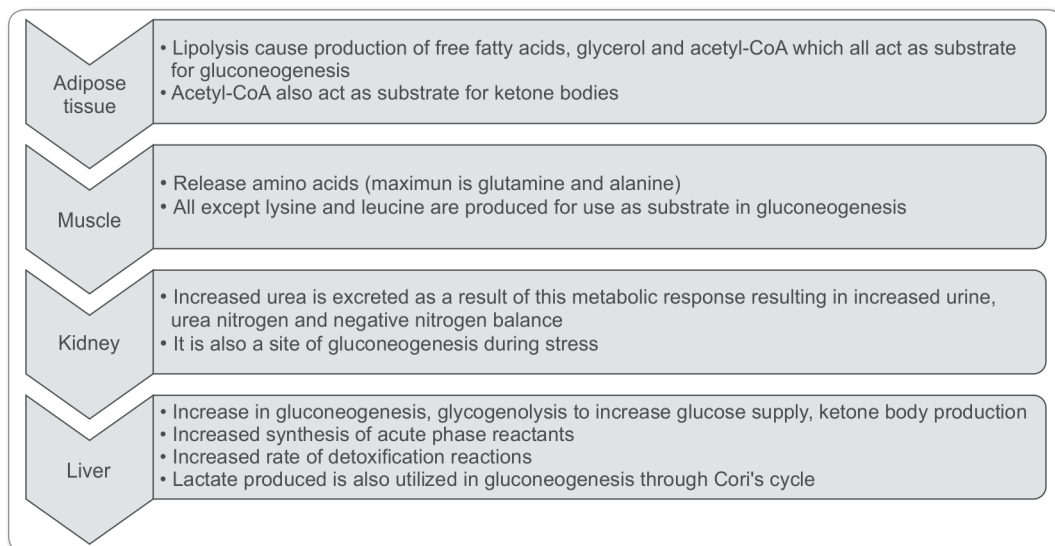
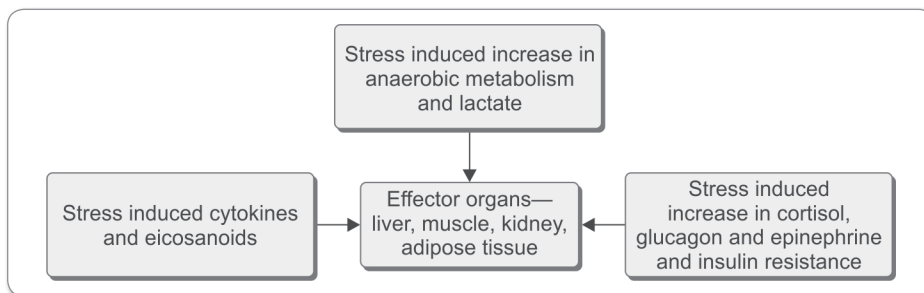
- Beta cells have alfa adrenergic receptors which on stimulation inhibit insulin release. Alfa receptors are stimulated in stress by beta endorphins, interleukin-1, glucagon and somatostatin
- On the other hand peripheral action of insulin is inhibited by cortisol, estrogen and progesterone. Insulin levels are decreased in ebb phase and increased in flow phase
- However, **stress is a state of insulin resistance, growth hormone resistance and thyroid deficiency**
- Alfa cells of islets have beta adrenergic receptors which are stimulatory. They are stimulated by hypoglycemia, stress, exercise and cause release of glucagon. Glucagon is increased in stress during both the ebb and the flow phase.

- **Ebb phase**—first 24 hours of stress when all metabolic activities slow down
  - Given by Cuthbertson
  - Main role is to conserve volume and energy
- **Flow phase**—has two divisions
  - Catabolic phase** during the first 3 to 10 days as followed by **anabolic phase** from 10 to 60 days after stress.
- During this phase, insulin/glucagon ratio is a better predictor of survival than either of them alone.
- Mineral response to stress—increase in copper levels and decrease in iron and zinc levels.

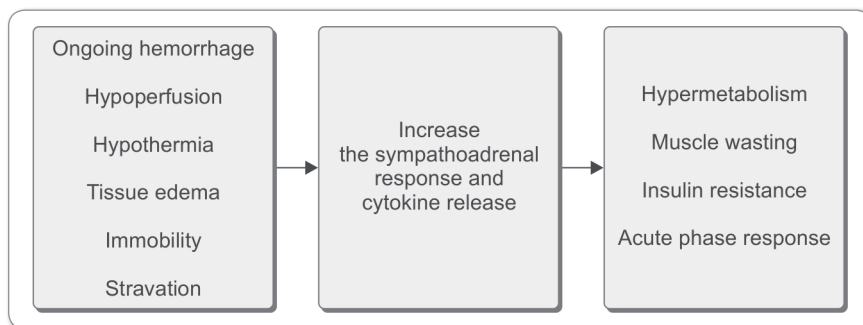
**Endocrine response to stress is as shown in the flow chart given below:**



**Metabolic response to stress is as shown in the charts below:**



**Factors affecting these responses to stress are as follows:**



**Q18. Write a note on nutritional assessment of surgical patient and estimation of his calorie needs.**

**Ans.**

**Nutritional assessment of a patient**

- **History**
  - Duration and amount of weight loss—weight loss > 10% in less than 6 months is significant
  - Decreased dietary intake
  - Gastrointestinal symptoms such as vomiting, diarrhea, anorexia
  - Other diseases, comorbid conditions and its relation to decreased nutrition.
- **Physical examination**
  - Subcutaneous fat
  - Pedal edema
  - Ascites
  - Muscle wasting
  - General physical examination from head to toe to look for nutritional deficiencies
- **Anthropometry**
  - Body mass index
  - Mid arm muscle circumference
  - Triceps skin fold thickness.
- **Laboratory analysis**
  - Serum protein and albumin—an albumin value between 3.5 to 5 mg/dL is adequate
  - Hemoglobin
  - Indirect calorimetry.

**Nutritional needs**

- **Basal energy expenditure** is given by Harris-Benedict equation and gives an estimate of calorie needs at basal metabolism level in an individual
- **Resting energy expenditure** is 10% greater than basal energy expenditure as it takes into account the work of breathing in addition to BEE. The resting energy expenditure is calculated using Weir equation  
3 REE values at 10% variation or 3 respiratory quotient values within 5% of each other is the REE of the patient
- **Total energy expenditure** is product of REE and stress factor. This stress factor is calculated using different stresses that a person is going on such as illness, shivering, food, physical activity and injury  
Total energy expenditure is the amount to be met by nutritional supplementation.
- **Normal protein requirement** is 0.8 to 1 g/kg/day and
- **Normal calorie requirement** is 25 to 30 kcal/kg/day for a person with moderate work and healthy state.

Other estimated calorie protein and stress factors are available for calculation of a specific patient.

**For example**, a patient with burns needs 35 to 40 kcal/kg/day calories and 2 to 2.5 g/kg/day of protein. The stress factor is 2.0 in these patients and so on for other patients with moderate or severe stress, trauma or surgery, the values linger between the above mentioned two extremes.



**Q19. Write a note on indications and methods of administering total parenteral nutrition.**

**Write a note on total parenteral nutrition therapy.**

**Ans.**

**Fundamental goals of nutritional support are**

- Meet the energy requirements for metabolic purposes
- Tissue repair
- Maintenance of core temperature
- Minimize the protein breakdown and preserve the lean body mass

**Indications of total parenteral nutrition**

- Seriously ill patients with severe malnutrition/sepsis/trauma when enteral feeding cannot be given
- Newborn with tracheoesophageal fistula, omphalocele, gastroschisis
- Infants with meconium ileus, short bowel syndrome
- High output enterocutaneous fistula
- Radiation enteritis/acute chemotherapy toxicity/postoperative ileus
- Weight loss preliminary to major surgery
- Cancer cachexia
- Ileus for > 10 days
- Patients with sprue, hypoproteinemia, pancreatic insufficiency
- Patients with esophageal dyskinesia after anorexia nervosa, cerebrovascular accident or psychogenic vomiting
- Patients with ulcerative colitis, regional enteritis or tuberculous enteritis
- Exacerbation of pancreatitis after enteral nutrition or pancreatitis with prolonged ileus.

**Contraindications**

- Hemodynamic instability
- Electrolyte imbalance
- When the prognosis does not support parenteral nutrition.

**Types of access**

- Peripheral parenteral nutrition can be used for < 2 weeks at a stretch and osmolality < 900 mosm/L through peripheral line.
- Central parenteral nutrition through central line.

**Constituents**

- Carbohydrate as 15 to 25% dextrose solution to provide 50 to 70% of the calorie requirements of the patient
- 3 to 5% crystalline amino acid solution. It is always to be administered after the glucose up to about 100 mg has been administered to avoid the utilization of the protein as an energy source
- Fat emulsion from soyabean or safflower oil to provide 15% of the total calories required by the patient
- Vitamin K is to be added as TPN formulations are deficient in vitamin K

- All other essential minerals and electrolytes are present in TPN
- Insulin is administered separately from the TPN bag as infusion or on sliding scale as per 6 hours glucose determinations.

### **Complications**

#### **• Complications related to the access site**

- Cardiac dysrhythmias
- Pneumothorax
- Catheter line infection
- Inadvertent arterial puncture
- Nerve or lymphatic injury
- Air embolism
- Venous perforation and hemothorax
- Venous thrombosis
- Catheter occlusion
- Catheter line infection or abscess

#### **• Metabolic complications**

- Hyperglycemia (most common) or hypoglycemia
- Hypertriglyceridemia
- Azotemia
- Osteoporosis
- Trace metal deficiency (**Zinc deficiency most common**)
- **Refeeding syndrome**
  - Patients with low BMI, increased unintentional weight loss and very low nutrient intake in recent past are at maximum risk
  - Occurs due to restitution of feeding at a very rapid rate after prolonged malnutrition which results in rapid working of the cellular pumps and resultant internalization of potassium, phosphorus and magnesium
  - This produces hypokalemia, hypomagnesemia and hypophosphatemia
  - Additional fluid load administered can result in congestive cardiac failure, cardiac arrhythmias and sudden death
- Liver dysfunction, fatty infiltration and steatosis
- Gallstones.

### **Q20. Write a note on enteral nutrition.**

#### **Ans. Fundamental goals of nutritional support are**

- Meet the energy requirements for metabolic purposes
- Tissue repair
- Maintenance of core temperature
- Minimize the protein breakdown and preserve the lean body mass

#### **Indications of enteral nutrition**

- Distal, low output, entrocuteaneous fistula
- Dysphagia except to liquids
- Patients after major trauma or surgery
- Protein energy malnutrition with poor oral intake

- To enhance adaptation after enterectomy
- Inflammatory bowel disease

**Contraindications**

- Severe pancreatitis
- Severe diarrhea
- Proximal or high output small intestinal fistula
- Small bowel obstruction or ileus

**Access**

- Nasogastric (Increased risk of aspiration)
- Nasoduodenal/nasojejunal route
- Percutaneous endoscopic gastrostomy/jejunostomy
- Surgical gastrostomy/jejunostomy

**Methods**

- Bolus or gravity method—250 to 500 mL 4–6 hourly
- Intermittent—some calculated amount hourly
- Continuous infusion—infusion at 20–40 mL/hour of full strength formula.

**Enteral nutrition formulas**

- Low residue isotonic formula with 1 kcal/mL is the first line formula
- Isotonic formula with dietary fiber (digestible dietary pectins)—delay intestinal transit and decrease the incidence of diarrhea.
- Immune enhancing formula—formulas containing branched chain amino acids, omega 3 fatty acids, glutamine, beta carotene or arginine
- Calorie dense formulas—2 kcal/mL for intragastric feeding
- High protein formulas—suitable for trauma/critically ill patients
- Elemental formulas—predigested nutrients with high osmolality. Need dilution before administration. Used in patients with malnutrition and pancreatitis
- Renal failure formula—contain essential amino acids and low volume
- Pulmonary failure formula—high fat content (50%) and low in carbohydrates
- Hepatic failure formula—increased branched chain amino acids.

**Complications***Metabolic complications*

- Hyperglycemia or hypoglycemia
- Hypertriglyceridemia
- Azotemia
- Osteoporosis
- **Refeeding syndrome**
  - Patients with low BMI, increased unintentional weight loss and very low nutrient intake in recent past are at maximum risk
  - Occurs due to restitution of feeding at a very rapid rate after prolonged malnutrition which results in rapid working of the cellular pumps and resultant internalisation of potassium, phosphorus and magnesium

- This produces hypokalemia, hypomagnesemia and hypophosphatemia
- Additional fluid load administered can result in congestive cardiac failure, cardiac arrhythmias and sudden death
- Liver dysfunction, fatty infiltration and steatosis
- Gallstones.

#### *Gastrointestinal complications*

- Abdominal cramps
- Abdominal distension
- Pneumatosis intestinalis and small bowel necrosis especially in the critically ill patients.

#### *Tube related complications*

- Displacement
- Blockage
- Perforation
- Infection
- Malposition.

## BLOOD TRANSFUSION AND DIC

**Q21. What is DIC (Disseminated intravascular coagulation)? Discuss its pathophysiology and management.**

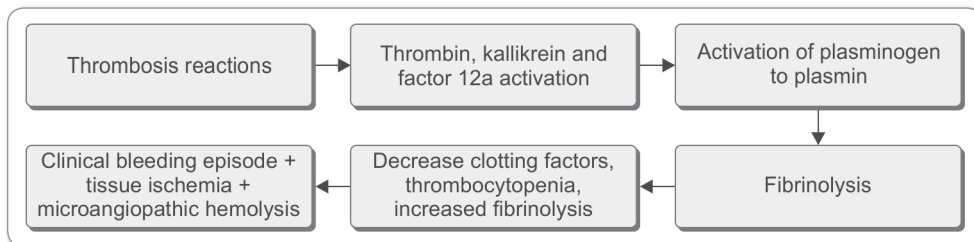
**Ans.** It is also called consumptive coagulopathy or consumptive thrombohemorrhagic disorder. It occurs because of uncontrolled activation of coagulation cascade at system wide level which leads to increased coagulation which leads to widespread thrombosis, resultant fibrinolysis which leads to depletion of clotting factor levels and thrombocytopenia with the final clinical result of bleeding.

#### **Pathophysiology**

##### 1. Activation of clotting cascade at a system wide level

<ul style="list-style-type: none"> <li>• Contact activation of factor 12 and intrinsic pathway</li> </ul>	<ul style="list-style-type: none"> <li>• Hemolytic uremic syndrome</li> <li>• Burns</li> <li>• Heat stroke</li> <li>• Glomerulonephritis</li> <li>• Endotoxins (gram negative sepsis, varicella, rickettsia)</li> <li>• Hemolytic transfusion reaction</li> </ul>
<ul style="list-style-type: none"> <li>• Activation of extrinsic pathway by provision of tissue factor</li> </ul>	<ul style="list-style-type: none"> <li>• Retained fetus</li> <li>• Placental abruption</li> <li>• Intrauterine fetal death</li> <li>• Amniotic fluid embolization</li> <li>• Massive trauma</li> <li>• Burns</li> <li>• Promyelocytic disorders</li> </ul>
<ul style="list-style-type: none"> <li>• Direct activation of factor 10, prothrombin, thrombin and factor 5.</li> </ul>	<ul style="list-style-type: none"> <li>• Snake venom</li> </ul>

2. Both the intrinsic and extrinsic pathways once activated by any of the above causative factors lead to activation of the clotting cascade which finally leads to increased intravascular coagulation and thrombosis
3. Thrombosis at a systemic level cause the following reactions:



### Clinical features

- Oozing from all IV line sites
- Petechiae and ecchymosis
- Gum bleeding, epistaxis, GI hemorrhage
- Shock, renal failure
- Intracranial bleeding and pulmonary hemorrhage
- Clinical features of underlying disease

### Investigations

- Thrombocytopenia
- Prolonged PT and aPTT
- Decreased fibrinogen and factor 8 levels
- Increased fibrin degradation products and D-dimer levels
- Fragmented RBCs on peripheral smear

### Management

- Management of underlying cause
- Volume resuscitation

Next line of therapies are used once underlying cause is corrected or in life threatening situations.

- Platelet transfusion to keep platelet  $>50,000/\text{cu mm}$
- Cryoprecipitate to keep fibrinogen level  $>100 \text{ mg/dL}$
- FFP to replenish clotting factor and  $\text{INR} <1.5$
- Indications of heparin therapy (to give always alongwith antithrombin 3 infusion)
  - Chronic DIC with no overt bleeding
  - When cause is amniotic fluid embolus
  - When cause is promyelocytic disorders
- EACA—given in hyperfibrinolytic state. Five gram loading dose is followed by  $1 \text{ g/hr}$  to block the fibrinolytic response.

**Results**

Despite all therapeutic measures, DIC has increased mortality because of both the DIC part and the underlying disease part.

**Q22. Write a note on Blood “substitutes”.**

**Ans.** Fluids that carry oxygen

**Ideal blood substitute has following features**

- Deliver oxygen
- No compatibility testing required
- Prolonged shelf life
- Cost effective
- Fewer side effects
- Persist in circulation

**Classes**

Biomimetic	Abiotic
Also called hemoglobin oxygen carriers (HbOC)	Also called perfluorocarbons (PFC) or nonhemoglobin oxygen carriers

**(HbOC)** – Three generations of HbOCs have been prepared but none have been approved for routine use by FDA.

*Problems with first generation HbOC include:*

- Osmotic diuretic effect
- Renal toxicity
- Short half life
- Coagulation abnormalities
- Vasoconstrictive properties because of scavenging of nitric oxide by free Hb

*Second generation HbOC:*

- They are pasteurized, compatibility testing free, long shelf and circulation life, no renal toxicity
- Include **DCLHb** (Diaspirin cross linked Hb) – made from outdated human blood Hb, polymerised bovine Hb – **HbOC – 201** and **Polyheme**—removes almost all tetrameric Hb and therefore thought to be more efficacious as because it has increased molecular weight, it has increased intravascular time
- Problems include free radical generation and exacerbation of reperfusion injury, methemoglobin production, immunosuppression and enhanced endotoxin pathogenicity
- Polyheme is associated with increased risk of myocardial infarction and therefore not FDA approved.

*Third generation HbOC*

- Liposomal HbOC
- Microsphere HbOC

- These have oxygen dissociation curve similar to RBC
- Still under research

### **PFCs (Abiotic)**

Dissolve more oxygen than plasma and therefore thought to be useful as blood substitutes.

#### *Problems*

- Immiscible in water and therefore need to be delivered as microdroplets
- Oxygen dissociation curve is linear and not sigmoid and therefore high  $\text{FiO}_2$  is required
- Need to be kept frozen.

#### *Second generation PFCs*

- Have increased oxygen carrying capacity
- Can be stored at 4°C
- Addition of lecithin as emulsifier has eliminated the adverse effects due to complement activation
- For example, **oxygen, oxyflour, oxycyte**
- Problems include increased incidence of vasoactive events mainly stroke
- Conclusion on their studies so far say that **“PFCs are not free of side effects and therefore not efficacious for oxygen delivery and use”**

**Q23. Enumerate the different blood components and write in brief about each of them.**

**Ans.**

#### **Whole banked blood**

- Upto 450 mL/donor
- Increase Hb by 1g/dl and hematocrit by 3 % per 1 bag whole blood
- Benefit is that it is metabolically more active and it is coagulation factor rich
- Disadvantage is that volume overload can occur

#### **Fresh whole blood**

- When transfused within 24 hours, it has shown improved outcomes in trauma associated coagulopathy.
- Provides greater coagulation factors levels.

#### **Packed RBCs (hematocrit is 60–70%)**

- Prepared by removing supernatant plasma after centrifugation
- Volume is 200–250 mL
- Preserved with using SAG-M (saline, adenine, glucose, mannitol) or CPDA (citrate, phosphate, dextrose, adenine), stored up to 35 days.
- Maximum half life of transfused RBC is 50 days (N – 120 days)

#### **Leucocyte reduced (filtration)/washed (saline) RBC**

- This process decreases HLA alloimmunization, febrile nonhemolytic transfusion reaction, decreases mortality due to infections (CMV) and decreases refractoriness to platelet transfusion.

**Fresh frozen plasma**

- Volume 200–250 mL
- Stored at  $-30^{\circ}\text{C}$
- First line therapy in fluid resuscitation in trauma associated coagulopathy in damage control resuscitation
- No rhesus antigen limitation
- Increases all factor levels by 2%
- Rich in fibrinogen (400 mg), protein C, protein S, antithrombin and plasma proteins.

**Cryoprecipitate**

- Supernatant of fibrinogen
- Rich in factor 8 (80 units) and fibrinogen
- Volume 15–17 mL
- Store at  $-40$  to  $-50^{\circ}\text{C}$

**Platelet concentrates**

- Volume 50 to 70 mL
- Stored with constant agitation up to 5 days at  $24^{\circ}\text{C}$
- Increases platelet by 5 to 10,000/unit
- Very useful for patients with bleeding disorders and in patients on clopidogrel therapy requiring surgery. It also require continuous infusion during procedure
- 6 random donor platelets = 1 apheresis platelet unit.

**Prothrombin complex concentrates**

- Factor 2, 9 and 10.
- Used mainly to treat warfarin overdose

**Autologous blood**

- Can be taken from patients planned for surgery with collection starting at around 40 days before surgery in patients with  $\text{Hb} > 11 \text{ g/dL}$  and hematocrit  $> 34\%$
- Collection is done at weekly interval with maximum 5 collections and last collection not less than 5 days before surgery.

**Indications of blood transfusion**

- Chronic anemia—in nonoperative patients who are not bleeding,  $\text{Hb} < 6 \text{ g/dL}$  is a definite indication. Exception—in cardiac patients,  $\text{Hb} < 8 \text{ g/dL}$  is a definite indication
- In perioperative patients and in patients with bleeding,  $\text{Hb} < 8 \text{ g/dL}$  is an indication
- In patients with acute blood loss  $> 20\%$  as a part of damage control resuscitation.

**Q24. Enumerate the complications of blood transfusion and discuss their management in brief.**

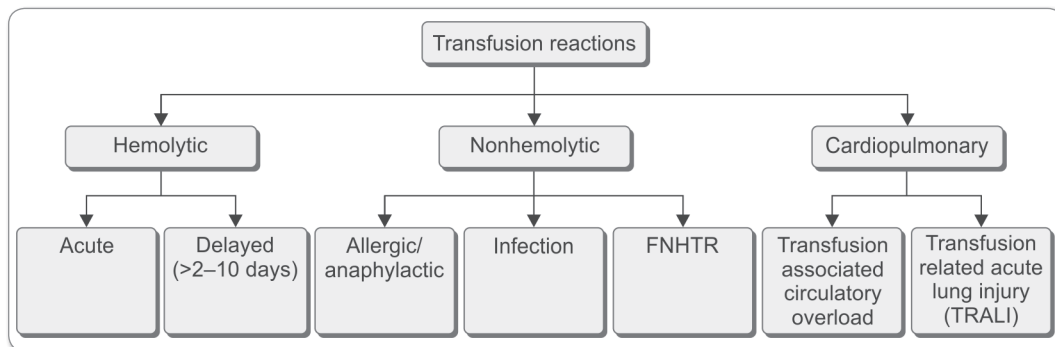
**Ans.**

- Incidence—10%
- Rate of infection transfer—HBV (30%) > HCV (3%) > HIV (0.3%)
- Most common cause—human error



- Most common reaction—febrile nonhemolytic transfusion reactions (FNHTR)
- Most lethal—TRALI > ABO incompatibility (hemolytic disorder) > bacterial contamination of platelets
- Most lethal infection—hepatitis.

**Classification** of reactions that can occur even with single transfusion



### Causes

#### FNHTR

- A form of graft versus host disease to transfused blood (GVHD)
- Preformed cytokines in donated blood and antibodies to donated blood

### Infections

Viruses	HCV, HBV, HGV, CMV HIV, HTLV, west nile virus
Prion	CJD (Creutzfeldt-Jakob disease)
Bacteria	Gram negative (RBC) Gram positive (Platelet) <i>Yersinia</i> , <i>Pseudomonas</i> , <i>Syphilis</i> , <i>Brucellosis</i> .
Parasites	<i>Plasmodium</i> , <i>Babesia</i> , <i>Ehrlichia</i> , Chagas disease

#### Allergy/anaphylaxis

- It is used for soluble transfusion of constituents

#### TACO (Transfusion associated circulatory overload)

- Large volume rapid transfusion

#### TRALI (Transfusion related acute lung injury)

- Anti—HLA antibodies or anti-HNA antibodies to pulmonary cells and leukocytes result in noncardiogenic pulmonary edema
- Onset is always within 6 hours.

#### Hemolytic

- Acute—preformed IgM to ABO antigen
- Delayed—preformed IgG to non ABO antigen

**Clinical presentation**

- Acute hemolytic reaction  
In awake—most common symptom is pain along intravenous site > flushing > back and chest pain  
most common sign is oliguria > hemoglobinuria  
In anesthetised—ongoing oozing and bleeding and hypotension
- Delayed Hemolytic reaction  
Indirect hyperbilirubinemia, anemia, elevated haptoglobin, positive Coomb's test
- Other symptoms—fever, chills, vomiting, diarrhea, rashes, itching, hives.

**Complications due to massive transfusion**

- Hypocalcemia
- Iron overload
- Hypothermia
- Coagulopathy
- Hypo/hyperkalemia
- Thrombocytopenia
- TACO
- Thrombophlebitis/Air embolism/DIC.

**Q25. Write a note on plasmapheresis.**

**Ans.** It is basically the removal, therapy and return/replacement of blood plasma into the blood circulation. This is also used as a method to collect plasma for industrial purpose. This is thus an extracorporeal therapy or segregation method.

**Procedure**

- Blood is initially taken out through a needle and plasma is removed by a cell separator which can be a discontinuous flow/ continuous flow centrifugation or plasma filtration
- After **plasma separation**, the blood cells are returned back into circulation while the plasma is treated to remove the antibodies or other disease producing substances and then returned into the circulation
- In **plasma exchange** therapy, the patient's plasma is discarded and replaced with replacement donor plasma, albumin or saline with added proteins
- Plasmapheresis just removes the products that are harmful. The disease treatment requires immunosuppression or other treatment to decrease the production of the substance or antibody.

**Applications***As therapy*

- Behcet's syndrome
- Chronic demyelinating polyneuropathy
- Guillain-Barre syndrome
- Refsum disease
- Myasthenia Gravis/Lambert-Eaton syndrome
- Thrombotic thrombocytopenic purpura/Hemolytic uremic syndrome

- HELLP syndrome, HIV Related neuropathy
- Antiphospholipid antibody syndrome and so on
- It is also used to reduce blood viscosity in diseases such as Waldenstrom macroglobulinemia, cyoglobulinemia, etc.

#### *As segregation process*

- Here the plasma donors are drained of blood similarly as in treatment and the plasma is separated from the blood and the donor red cells are returned back to circulation. This allows nearly up to 1 liter of plasma to be donated at a time and also frequent (weekly) donation
- The plasma can then be used as direct plasma transfusion or segregation into factors, albumin, immunoglobulin or fresh frozen plasma transfusion.

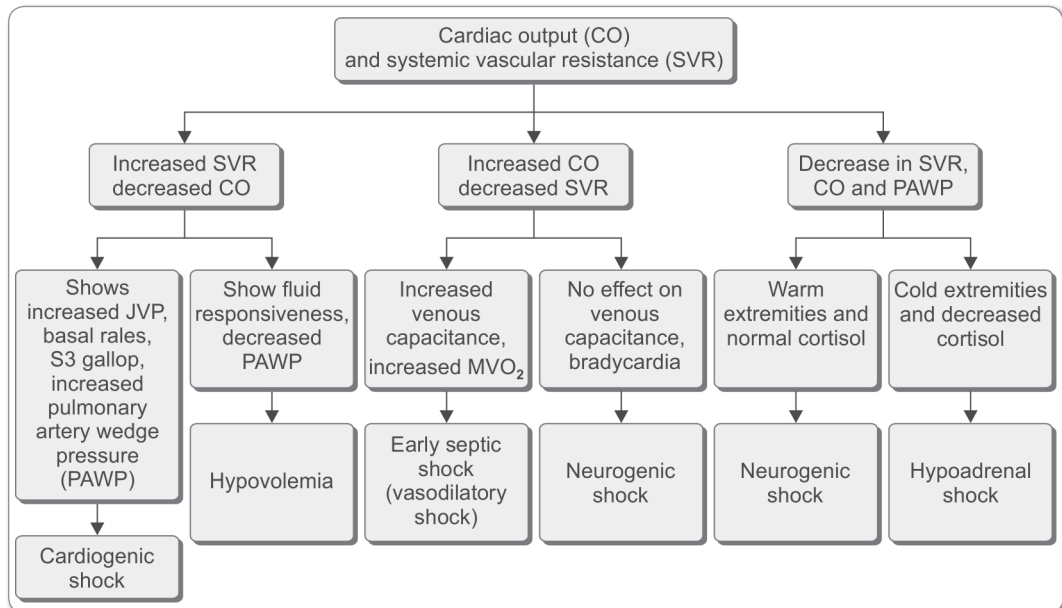
#### **Complications**

- Bleeding at the catheter site, infection, hematoma
- Risk of transfusion reactions
- Citrate toxicity and hypocalcemia
- Immunosuppression and increased susceptibility to infections
- Transfusion transmitted infectious diseases.

## **SIRS, SHOCK AND MODS**

**Q26. Write a note on the classification and differential diagnoses of shock in surgery.**

**Ans.**



- Blalock's etiological classification of shock

<b>Oligemic shock</b>	Hematologic causes (blood loss)
<b>Loss of ECF volume (hypovolemic shock)</b>	Vomiting, diarrhea, fistula
<b>Neurogenic shock</b>	Spinal shock
<b>Cardiogenic shock</b>	Extrinsic—cardiac tamponade, pneumothorax Intrinsic—myocardial infarction
<b>Vasogenic shock</b>	Vascular dilation as in sepsis

### Differential diagnoses of shock types

Important terms while diagnosing shock

- Cardiac index = cardiac output/body surface area [CI = CO/BSA]
- Cardiac output = heart rate stroke volume [CO = HR SV]
- Blood pressure = Cardiac output systemic vascular resistance [BP= CO SVR]
- Pulse pressure = systolic – diastolic pressure
- Mean arterial pressure (MAP) = (Systolic BP + 2 diastolic BP)/3
- Cerebral perfusion pressure (CPP) = MAP – ICP/CVP whichever is greater
- Heart rate > 120, SBP < 90 mm Hg or > 40 mm Hg decrease, MAP < 65 mm Hg are all markers of shock.

### Q27. Discuss the pathophysiology of septic shock and MODS (multiple organ dysfunction syndrome).

**What is MODS? Discuss how it develops in the patients.**

Ans.

#### Important definitions

- **Infection**  
A response to the presence of microorganisms in body
- **Bacteremia**  
The presence of viable bacteria in circulating blood
- **Systemic inflammatory response syndrome (SIRS)**  
The systemic inflammatory response to a wide variety of severe clinical insults, manifested by two or more of the following conditions:
  - Temperature > 38°C or < 36°C
  - Heart rate > 90 /min
  - Respiratory rate > 20 /min or PaCO<sub>2</sub> < 32 mm Hg
  - WBC count > 12,000/mm<sup>3</sup>, < 4000/mm<sup>3</sup> or > 10% band forms.
- **Sepsis**  
SIRS + infection
- **Severe sepsis**  
Sepsis associated with organ dysfunction, hypoperfusion or hypotension.
- **Refractory septic shock**  
Sepsis induced hypotension despite adequate fluid resuscitation
- **Multiple organ dysfunction syndrome (MODS)**  
Presence of altered organ function (2 or more organ systems) in an acutely ill patient

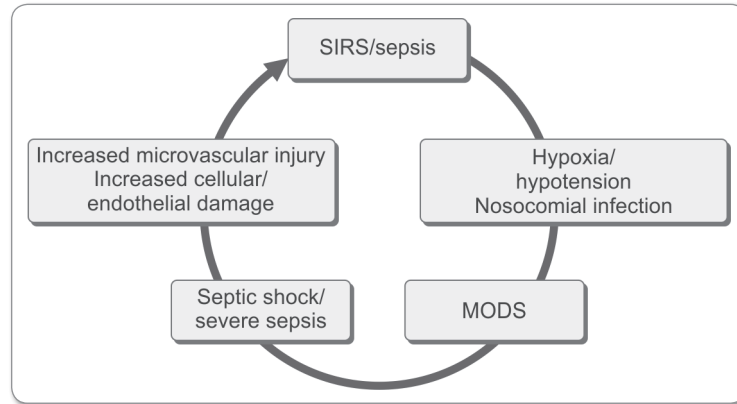
### Pathophysiology

1. An **infectious insult**, e.g. pneumonia, UTI, perforated viscus, necrotizing fasciitis, etc. or a **noninfectious insult** (Burns, trauma, pancreatitis, ruptured abdominal aortic aneurysm, etc.) triggers local inflammation and/or ischemia which is characterized by a cellular and a vascular response as follows:

Cellular response	Vascular response
Increase in TNF leads to <ul style="list-style-type: none"> <li>• Muscle catabolism</li> <li>• Increase PAF, IL-1, 6, prostaglandins and steroids</li> <li>• Increase expression of adhesion molecules</li> <li>• Increased neutrophil function and</li> <li>• Increased free radical production</li> </ul>	<ul style="list-style-type: none"> <li>• Increased tissue factor</li> </ul>
	<ul style="list-style-type: none"> <li>• Increased TNF cause increased PAI-1 (plasminogen activator inhibitor-1)</li> </ul>
	<ul style="list-style-type: none"> <li>• Decrease in protein C levels because of increased alpha-1 antitrypsin levels</li> </ul>
	<ul style="list-style-type: none"> <li>• Decreased antithrombin 3 and tissue factor pathway inhibitor levels thus resulting in a <b>Procoagulant state</b></li> </ul>

2. Now, the further effects depend on the body's response to these two events. If CARS (compensatory anti-inflammatory response system) predominates, then inflammation is controlled once the trigger is taken care of. However, if systemic inflammatory response syndrome (SIRS) predominates in an uncontrolled way, the further events continue as follows.
3. Because of increased inflammation and a procoagulant state, end organ ischemia results. This causes lactic acidosis and decreases ATP level which leads to increased intracellular calcium followed by increased and uncontrolled activation of intracellular proteases.
4. These proteases mediate the next event in cell injury
  - Normally, in presence of adequate oxygen, hypoxanthine is converted to xanthine and then to uric acid in cells by xanthine dehydrogenase
  - However, in presence of activated proteases, xanthine dehydrogenase gets converted to xanthine oxidase
  - When reperfusion occurs in presence of oxygen, it leads to conversion of hypoxanthine to uric acid by xanthine oxidase which produces superoxide radicals as a byproduct
  - These superoxide radicals also further leads to production of hydroxyl radicals and singlet oxygen
  - These free radicals cause peroxidation of lipid bilayer of cell and cause cell membrane damage, therefore, causing ischemia reperfusion injury
5. This leads to increased capillary leaks, activation of inflammatory cascade and increased nitric oxide production which leads to hyperpolarization of plasma membrane and is responsible for lipopolysaccharide induced refractory hypotension and vasodilatation seen in severe sepsis
6. Again ineffective CARS and overactive SIRS leads to ongoing increased endothelial and cellular damage as well as ongoing ischemia reperfusion injury at cellular level which leads to SIRS/sepsis.

**This results in a deadly vicious cycle as follows**



This vicious cycle leads to MODS which can only be managed with appropriate and timely treatment measures and if patient responds to these treatment measures.

Benefit from treatment—recovery

Unresponsive patient in MODS—death.

**Q28. Discuss the management guidelines for sever sepsis/septic shock.**

**How will you investigate a patient in sepsis? Outline its management.**

**Ans.** Definitions as mentioned in question “Pathophysiology of septic shock and MODS (multiple organ dysfunction syndrome)”

#### Investigations

<b>Immediate</b>	<ul style="list-style-type: none"> <li>• Arterial blood gas analysis</li> <li>• CVP (N. 8–12 mm Hg)</li> <li>• MAP (&gt; 64 mm Hg)</li> <li>• Hb (&gt; or = 7), platelet (&gt; 1,00,000/cu mm), TLC, DLC</li> <li>• C-reactive protein, procalcitonin levels</li> <li>• Blood lactate levels (&lt; 1 mmol/L)</li> <li>• Mixed venous oxygen saturation &lt; 65%, <math>SVCO_2</math> &lt; 70%</li> </ul>
<b>Within 45 minutes</b>	Blood culture All indwelling lines cultures Imaging studies for possible site of infection 1-3 beta-d glucan levels Mannan and anti-mannan antibody levels.

#### Initial resuscitation

- Crystalloids at 30 mL/kg bolus (called fluid challenge). Apart of this can be albumin
- Blood if Hb < 7g/dL or hematocrit < 24%
- Vasopressors—target MAP (mean arterial pressure) > 64 mm Hg norepinephrine (DOC). If patient does not respond to both fluid challenge and nor-epinephrine, consider starting epinephrine and last resort should be dopamine.

Dopamine is not the preferred vasopressor because:

- It causes more tachycardia
- Is arrhythmogenic
- Is immunosuppressive
- Causes endocrine manipulation due to HPA axis interaction.
- Platelet transfusion if
  - Counts < 10,000/ cu mm
  - Patient going to surgery with platelet <50,000/cu mm
  - Bleeding patient with platelet <20,000/cu mm
- Glycemic control to be established
- Steroid can be given as the last ditch support. 200 mg/day continuous infusion of steroid is better than bolus infusion
- If still no response to vasopressors and inotropic support required, inotrope of choice in septic shock is dobutamine.

#### **Other measures**

- Antibiotics—start empiric broad spectrum antibiotics within 1 hour of patient treatment. Deescalate once the cultures are available
- Nutrition—enteral nutrition is preferred and immune enhancing formulas can be used to hasten recovery
- DVT and stress ulcer prophylaxis to be considered
- Patient should receive ICU care with noninvasive or invasive ventilation and sedation, analgesia, muscle relaxants as per requirement
- Care to avoid the occurrence of bedsores.
- There is no role of hydroxyethyl starch or other volume expanders, selenium, immunoglobulins, erythropoietin, activated protein C, bicarbonate (unless when pH < 7.15), renal dose dopamine, bolus steroid in absence of shock or specific indication.

### **Q29. Write a note on pathophysiology of hemorrhagic shock.**

#### **Discuss the pathophysiology of lethal triad.**

**Ans.** The hemorrhagic shock is classified as follows:

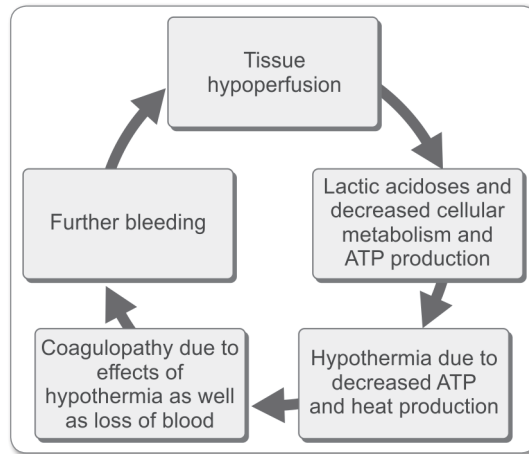
- **Class I**—Up to 15% blood loss (750 mL). Manifest only as mild anxiety.
- **Class II**—Up to 30% blood loss (1500 mL). Manifest as tachycardia and change in pulse pressure.
- **Class III**—Up to 40% blood loss (2000 mL). Manifest as hypotension besides above changes.
- **Class IV**—More than 40% blood loss (> 2000 mL).

The components of lethal triad are:

- Hypothermia
- Acidoses
- Coagulopathy

It is called so because this is a vicious cycle which is difficult to contain and once the patient develops it usually culminates in death of the patient.

The pathophysiology of hemorrhagic shock is basically the result of tissue hypoperfusion and it leads to lethal triad as follows:



As shown in the cycle above, decreased tissue perfusion leads to decreased cellular metabolism, decreased ATP production and increased lactate production due to anaerobic metabolism as well as due to decreased clearance of metabolites. This produces hypothermia.

### Effects of hypothermia

- Increased fibrinolytic activity
- Decreased thromboxane formation and resultant platelet adhesion function
- Decreased enzyme activity responsible for coagulation
- Decrease in the synthesis of Hageman factor (XII) and thromboplastin
- It can lead to production of heparin-like substance and lead to DIC like syndrome.

All of which finally leads to coagulopathy

- Hypothermia for prolonged period also leads to slowing of the metabolism rate at cellular level and can lead to exacerbation of acidoses.

### Other causes of coagulopathy

- Dilutional cause: Due to loss of blood and alongwith it the important clotting factors and replacement by fluids or stored blood
- Consumption coagulopathy.

**Effects:** Propagation of lethal triad due to ongoing bleeding and further coagulopathy

**The method to prevent or circumvent the lethal triad** is by damage control resuscitation as explained in the question on damage control in surgery



## BASIC OF SURGICAL TECHNOLOGIES AND ADVANCED SURGERY

### Q30. What is minimal access surgery? Enumerate its various techniques.

**Ans.** Minimally invasive surgery is the operation performed through minimal possible physical, physiological and psychological trauma.

The evolution of technology and its application to surgery has made this possible.

The different techniques under the domain of minimally invasive surgery are as follows:

- Laparoscopy
- Thoracoscopy
- Retroperitoneoscopy
- Mediastinoscopy
- Endoscopy—ERCP, colonoscopy, UGI endoscopy, bronchoscopy, cystoscopy, sigmoidoscopy, endovascular surgery, arthroscopy and so on.
- Natural orifice transluminal endoscopic surgery (NOTES)
- Single incision laparoscopic surgery (SILS) or single port surgery (SPS)
- Robotic surgery

### Q31. Write a note on advantages of laparoscopy.

**Write a note on drawbacks of laparoscopy.**

**Write a note on complications of laparoscopy.**

**Write a note on the physiological changes produced by CO<sub>2</sub> pneumoperitoneum.**

**Ans.** Laparoscopy is minimally invasive procedure performed by instilling CO<sub>2</sub> into the peritoneal cavity and using ports and instruments to carry the procedure out through small holes.

The **physiological changes** produced by CO<sub>2</sub> pneumoperitoneum are as follows:

<b>Ventilation changes</b>	<ul style="list-style-type: none"> <li>• Decrease in compliance</li> <li>• Decreased functional residual capacity</li> <li>• Increased PaCO<sub>2</sub>, VCO<sub>2</sub>, End tidal CO<sub>2</sub>—these changes plateau after about 30 minutes of starting the procedure</li> <li>• Dead space and shunt fractions remain constant</li> </ul>
<b>Cardiovascular changes</b>	<ul style="list-style-type: none"> <li>• Decreased venous return and cardiac output</li> <li>• Increased systemic and pulmonary vascular resistances</li> <li>• Increased arterial pressures, increased cardiac filling pressures, increased afterload</li> <li>• The heart rate remains bradycardic initially but then normalises (<b>Bradycardia is the most common arrhythmia</b> during pneumoperitoneum)</li> </ul>
<b>Respiratory changes</b>	<ul style="list-style-type: none"> <li>• Subcutaneous emphysema</li> <li>• Pneumothorax, pneumomediastinum—spontaneous resolution in 30 to 60 minutes</li> <li>• Migration of endotracheal tube and endobronchial intubation</li> <li>• Gas embolism</li> <li>• Hypercarbia with respiratory acidosis</li> </ul>

*Contd...*

*Contd...*

<b>Renal changes</b>	<ul style="list-style-type: none"> <li>Decrease in renal plasma flow, glomerular filtration rate, urine output</li> </ul>
<b>Hypercoaguable state</b>	<ul style="list-style-type: none"> <li>Pneumoperitonum and reverse trendelenburg position that is used both increase venous stasis in lower limbs. Patients of old age, obesity, prolonged surgery are at increased risk</li> <li>A Caprini score of 2 is given to all laparoscopic surgeries &gt; 45 minutes and mechanical prophylaxis is advised in them</li> </ul>
<b>Stress and endocrine responses</b>	<ul style="list-style-type: none"> <li>Cortisol elevation is more in laparoscopy than in open surgeries</li> <li>However, stress induced changes such as cytokine elevation, leucocytosis, hyperglycemia, immune system changes are less in laparoscopy compared to open surgery.</li> </ul>

### **Advantages of laparoscopy are as follows:**

#### *Patient*

- Rapid postoperative recovery and early return to work
- No scar and therefore no scar related complications such as pain, incisional hernia, wound infection, seromas, hematoma, dehiscence or scar tenderness
- Aesthetic value due to no scar which also gives psychological advantage.

#### *Operation*

- Less need of blood transfusion during surgery due to less blood loss
- Less need of analgesics and antibiotics
- Less chances of adhesion formation and visceral exposure therefore, less chances of infection transmission.

#### *Hospital*

- Rapid turnover of patients so more economical gain
- Shorter hospital stay and less complications improve the hospital reviews
- Laparoscopy is more educational than open surgery as all can view the television and learn. So it is a better teaching tool for the hospitals.

### **Disadvantages of laparoscopy**

- Loss of natural hand eye coordination as the camera (eyes of laparoscope) is in the hands of the assistant
- Loss of 3 dimensional visual perception—the perception of depth is impaired. However, this is being taken care of by 3-D systems that are now available
- As the instruments have fixed motion range at trocar points, the range of motion is limited
- There is loss of tactile feedback as the surgeon cannot feel the structures directly
- Exaggeration of physiological tremors and compromised ergonomics
- This leads to increased surgeon and assistant fatigue
- Has longer learning curve. Due to loss of wrist like motion, suturing and knot-tying are particularly difficult
- Procedure itself is more costly and may take longer time than open procedure. This, however, can be taken care of by decrease in the postoperative costs.

## Complications of laparoscopy

### Injuries

<b>Verres needle injuries</b>	<ul style="list-style-type: none"> <li>• Usually not significant because of the small diameter of Verres needle</li> <li>• Can be visceral or vascular injury which is taken care of after trocar placement</li> </ul>
<b>Trocar injuries</b>	<ul style="list-style-type: none"> <li>• Inferior epigastric artery bleed</li> <li>• Small intestine or large intestine perforation</li> <li>• Solid visceral injury</li> <li>• Urinary bladder injury</li> <li>• Major mesenteric or retroperitoneal vessel bleed</li> </ul>
<b>Instruments related injury</b>	<ul style="list-style-type: none"> <li>• Injury due to insulation failure or capacitive coupling (Described in question on electrocautery) can lead to visceral burns and delayed perforation peritonitis (after 4 to 5 days) or cutaneous burns.</li> <li>• Injuries by misidentification (e.g. common bile duct injury during laparoscopic cholecystectomy)</li> <li>• Injuries during manipulation and retraction—bowel injuries, liver injuries, etc.</li> <li>• Injuries during dissection and hemostasis to any organ</li> <li>• Tears and splits in organ while retrieval or handling (e.g. gallbladder perforation, spleen rupture, etc.)</li> </ul>

### Pneumoperitoneum

- Cardiac arrhythmias—bradycardia, ectopics, asystole
- Gas embolism
- Pneumothorax, pneumomediastinum
- Hypothermia and peritoneal trauma due to cold CO<sub>2</sub> gas exposed during insufflations.

### Postoperative complications

- Though the risk is less, there is still chance of port site infection and abscess
- Delayed risk of port site hernia
- Port site metastasis if the laparoscopy was performed in malignancy cases
- Deep vein thrombosis of lower limb
- Basal atelectasis and pneumonia
- Shoulder tip pain due to CO<sub>2</sub> causing phrenic nerve irritation

## Contraindications of laparoscopy

Though there are no definite absolute contraindications or definite guidelines on these issues, the following are routinely considered as contraindications to laparoscopic surgeries and are listed below:

### Absolute

- Third trimester of pregnancy—lower abdominal and pelvic surgeries by laparoscopy are especially not to be done
- Child C liver disease or liver disease with gross ascites
- Patients with hemorrhagic shock and hemodynamic instability

- Patients with severe cardiopulmonary compromise such as severe COPD or severe congestive heart failure.

#### *Relative*

- Locally invasive malignancies—though laparoscopy is being used in early stage malignancies, locally invasive malignancies are still considered contraindication to laparoscopy because of the risks of cancer dissemination and/or port site metastasis. Also malignancies such as mucinous or signet cell adenocarcinomas are friable, prone to disseminate and implant and are therefore considered relative contraindication
- Intraperitoneal mesh placed in the area of surgery
- Multiple previous intraperitoneal surgeries in the same area
- Inexperience
- Uncorrected coagulopathy is a relative contraindication
- Patients with acute brain injury and ventriculoperitoneal shunts are also relative contraindication.

### **Q32. Enumerate the indications of diagnostic laparoscopy**

**Ans.** The broad categories and indications of diagnostic laparoscopy are as follows:

<b>To ascertain the cause of abdominal pain</b>	<ul style="list-style-type: none"> <li>• Peritonitis</li> <li>• Gastrointestinal hemorrhage of unexplained etiology</li> <li>• Intussusception</li> <li>• Postoperative adhesions</li> <li>• Mesenteric ischemia</li> <li>• Gynecologic causes</li> <li>• Cases of small bowel obstruction</li> </ul>
<b>In blunt or penetrating trauma</b>	<ul style="list-style-type: none"> <li>• To rule out diaphragmatic injuries</li> <li>• Exclude peritoneal breach in penetrating trauma</li> </ul>
<b>In abdominal or pelvic tumors</b>	<ul style="list-style-type: none"> <li>• Confirmation of disease (Peritoneal carcinomatosis)</li> <li>• Staging laparoscopy</li> </ul>
<b>In Infectious causes</b>	<ul style="list-style-type: none"> <li>• Acalculous cholecystitis</li> <li>• Ruptured liver abscess</li> </ul>

### **Q33. Write a note on NOTES (Natural orifice transluminal endosurgery).**

**Ans.**

- Natural orifice transluminal endoscopic surgery (NOTES) is an emerging experimental alternative to conventional surgery that eliminates abdominal incisions and incision-related complications by combining endoscopic and laparoscopic techniques to diagnose and treat abdominal pathology
- The SAGES (Society of American Gastrointestinal Endoscopic Surgeons) and ASGE (American Society of Gastrointestinal Endoscopy) came together in 2005 to establish NOSCART (natural orifice surgery consortium for assessment and research). They gave the term NOTES
- In NOTES, the operation is performed in the peritoneal cavity having gained access through a natural orifice rather than a direct transcutaneous access
- In hybrid NOTES, the operation NOTES is combined with some part of operation being performed through direct transcutaneous access.

**Technique***Peritoneal access*

- Should be safe
- Minimal tissue injury
- Good exposure
- Ability to manipulate instruments and at the same time have a good seal around the instrument.

*Options*

- PEG approach (transoral and through stomach)
- Transvaginal
- Transrectal.

*Closure*

- Apparatus for closure are available such as T tags, star tags, basket tags, etc.
- This is an area of surgery still under research.

*Procedures contemplated*

- Tubal ligation
- Appendicectomy
- Splenectomy
- Oophorectomy
- Cholecystectomy—first transvaginal cholecystectomy was performed by Marescaux and Bessler (2007).

**Challenges**

- **Intraperitoneal complications**
  - Early or delayed presentation
  - Can lead to infection, bleeding, anastomotic leak, closure site dehiscence
  - Vascular injury during access procedure
  - Damage to viscera
  - Adhesion formation
  - Post NOTES dyspareunia
- **Dexterity and technological challenges**
  - Requisition of a multichannel NOTES platform that can bend in more than 2 axes
  - Present available instruments are not robust enough to provide the much required properties of triangulation, retraction and dissection
  - Spatial orientation of surgeons to endoscopic views and of endoscopists to the laparoscopic view requires training
  - Instruments currently available are not capable of providing good suturing in NOTES whether for access site closure or intraoperative suturing for hemostasis control or anastomosis.
- **Training, education and availability of the equipment is yet another problem.**

**Benefits of NOTES**

- Absence of incisional complications including pain, hernias and external wound infections
- Hopefully there would be fewer adhesions
- A decreased need for anesthesia
- A shorter hospital stay

**Q34. Enumerate the uses of therapeutic embolisation.**

**Ans.** The development of techniques for inserting a catheter percutaneously and manoeuvring it into almost any artery with safety and confidence is one of the major advances in medicine. At first the main aim was to obtain better angiograms, but now arterial catheterisation has acquired increasing therapeutic value. In addition to allowing regional infusions of drugs and mechanical dilatation of stenoses in arteries, catheters may now be the route by which the radiologist deliberately introduces emboli into vessels feeding tumours, vascular abnormalities or sites of bleeding. This approach is called therapeutic embolization.

**Indications**

- Vascular malformations: Occlusion of congenital or acquired aneurysms (cerebral, visceral, extremities), pseudoaneurysms, vascular malformations or other vascular abnormalities that have potential to cause adverse health effects
- Nontraumatic hemorrhage: Treatment of acute or recurrent hemorrhage (e.g. hemoptysis, gastrointestinal bleeding, postpartum and iatrogenic hemorrhage and hemorrhagic neoplasms)
- Trauma: For control of dramatic hemorrhage, for example, related to splenic laceration or pelvic fractures
- Uterine artery embolization: Devascularization of benign uterine leiomyomas and adenomyosis for symptom alleviation or to reduce operative blood loss
- Oncologic embolization: To relieve symptoms, prevent or treat hemorrhage, reduce operative blood loss or improve survival and quality of life. For example primary and secondary hepatic malignancies, renal cell carcinoma and primary and secondary bone malignancies
- Tissue ablation: Ablation of benign neoplastic and nonneoplastic tissue that produces adverse health effects to the patient (e.g. hypersplenism, refractory renovascular hypertension, untreatable urine leak, proteinuria in end-stage kidney disease, renal angiomyolipoma, varicocele, pelvic congestion syndrome, priapism, and abdominal pregnancy).
- Flow redistribution:
  - To protect normal tissue (e.g. gastroduodenal artery and right gastric artery embolization in hepatic artery chemoembolization and radioembolization or
  - Proximal superior gluteal artery coil embolization during particle embolization of the anterior division of the internal iliac artery for tumor devascularization) or
  - To facilitate other subsequent treatments (e.g. right portal vein embolization to induce left lobe hypertrophy before surgical resection).

- Endoleak management: Including direct sac puncture or collateral vessel embolization for endoleaks
- Regional therapy delivery: Vehicle for delivery of drugs or other agents that may include oncolytic viruses, chemotherapy, beta -emitting spheres or other agents used to treat an organ or specific target lesion
- Enterocutaneous tracts and lymphatic abnormalities: Embolizing abnormal communications between organs from cavities or organs to the skin surface, thoracic duct leaks, lymphedema.

**Embolisation materials include** autologous clot, gelfoam, muscle, silastic balls, silicone balloons, steel coils and cyanoacrylic glue.

Some produce permanent occlusions, some temporary.

### Q35. Write a note on surgical diathermy.

**Write a note on electrocautery uses in surgery.**

**Ans.** Electrocautery uses direct current whereas electrosurgery uses alternating current

#### Principles of thermal tissue destruction

- 60°C—coagulation necrosis
- 80°C—carbonization and shrinkage
- 100°C—cell vaporization—gas and smoke
- >100°C—carbon residue/Eschar formation

Current flows from electrosurgical unit to patient via active electrode and returns to unit via return electrode.

#### Types

- **Monopolar**—return electrode is through the patient (grounding plate) and therefore path of current is unpredictable
- **Bipolar**—current passes between electrodes and flow of current beyond surgical field is minimal. It is approved for sealing vessels up to 7 mm in diameter.

#### Current effects

Cutting	Coagulation	Fulguration
Continuous wave	Pulsed waveform	High power
Low voltage	High voltage	Low current density
High frequency	Low frequency	

However, coagulating current can be used as cutting current by decreasing the surface area of contact between active electrode and skin and therefore increasing current density.

**For example**, in laparoscopy the use of monopolar electrosurgery by “L” hook shows that when the tip of L hook is used, it decreases the surface area and acts as cutting electrode while the base of L hook acts as coagulating electrode by decreasing current density with the same current flow.

**Fulguration** is produced when low current density and high power current by outer side of L or spatula is used at an increased distance (no touch technique) from the specific bleeding point which allows superficial tissue heating.

### Special issues with laparoscopic electrosurgery

#### Insulation failure

- Laparoscopic instruments are normally insulated up to their tip. When this gets removed, injury to adjacent viscus can occur or current can pass through metal trocar and dissipate unknowingly causing injury
- The effect of insulation failure can thus be outside of the visual field of laparoscopy and may present late.

#### Direct coupling

- When one conducting material touches or arcs to another one deliberately or inadvertently, there is transfer of current from the conducting electrode to the touching electrode. This is called direct coupling.

#### Capacitive coupling

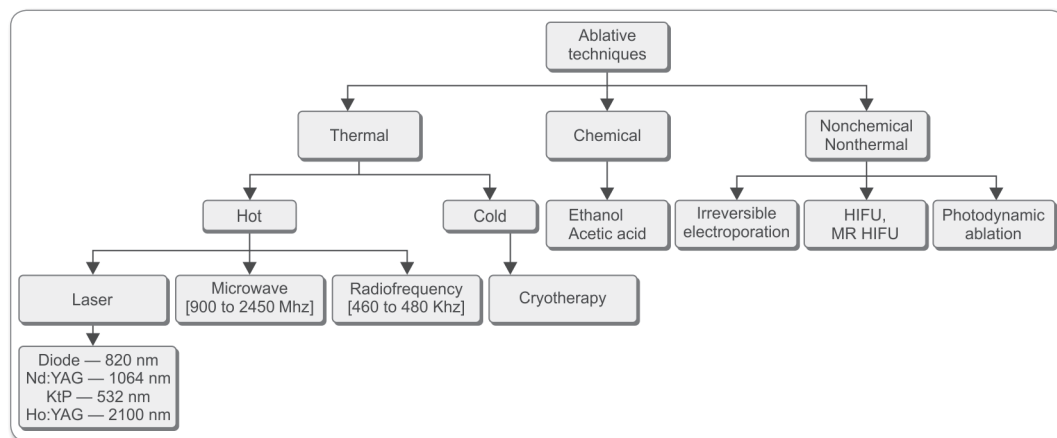
- Conductor has intact insulator but, passes through a non-insulated conductor such as a metal trocar or metal suction tip
- This specifically occurs when using activation of electrode while not in tissue contact or during fulguration
- This can be avoided by using all metal trocars as a large conductive surface area dissipating energy over a wide area.

### Some interesting points to know in laparoscopy

- **Verres needle**
  - Maximum flow is 2.5 L/min
  - 14 gauge
  - External diameter 2 mm
  - Length is 70–120 mm
- **Hasson cannula's** maximum flow is >6 L/min
- Frequency of **harmonic scalpel** is 55,500 Hz
- Frequency of **CUSA** is 23,000 Hz

### Q36. Classify the ablative techniques used in surgery.

Ans.





**Q37. What is Photodynamic therapy? Enumerate its principles and application.****Ans.**

- Also called photochemotherapy
- It involves the use of photochemical reactions through the interaction of light, oxygen and photosensitising agents.

**Mechanism of action**

- Usually a two step procedure.
  - **First step**—administration of photosensitizer through topical/oral/intravenous route which is taken up by target cells and gets collected usually in the vicinity of the mitochondria
  - **Second step**—activation of the photosensitizer in presence of oxygen with help of a specific wavelength of light either blue (405 to 420 nm called **sorret band**) or red (635 nm)
- In skin, the photosensitizers mainly accumulate in the sebaceous glands and the epidermal cells
- The activation of these photosensitizers causes activation of the reactive oxygen species and emission of light which is called photofluorescence
- Singlet oxygen species which are believed to be type 2 photochemical reaction are seen to predominate in this therapy
- Other mechanisms of action
  - Modify cytokine expression
  - Increase interleukin-1 beta, interleukin 2, TNF-alfa and G- CSF
- Photosensitizing agents include aminoleulinic acid or methyl aminolevulinate. Others include porphyrins, xanthenes, phenothiazines, monoterpenes and chlorines dyes.

**Indications**

- Actinic keratosis
- Small basal cell carcinomas
- Bowen's disease
- Acne
- Photoaging
- Vitiligo, psoriasis, neurodermatitis, eczema, cutaneous T-cell lymphoma and lichen ruber planus
- Wet age related macular degeneration
- Carcinoma esophagus
- Cholangiocarcinoma.

Recent addition of laser photodynamic therapy wherein laser is used to initiate the photochemical reaction has been added to this field because monochromaticity of laser provides the maximum effectiveness amongst all the various light sources used.

**Q38. Write a note on day case surgery.**

**Ans.** Day case surgery is when a patient gets his procedure from first consultation to **discharge after admission within 12 hours** of the hospital stay.

- 12 hours to 24 hours is **overnight stay**
- 24 hours to 72 hours is **short stay surgery**

- This depends on appropriate patient and procedure selection for day case surgery. Also the patient should have appropriate support system to take his care of once discharged.

**Selection parameters**

- Patients up to ASA III are considered for day case surgery
- Patients should have a good support system and home quite near to the facility with good and safe transport facility to bring him back in case the need arises
- Procedures should not be longer than 2 hours and minimally invasive procedures are preferred.

**Steps**

Patient selection and procedure selection.

*Preoperative assessment*

- History and physical assessment
- Medication and current comorbidity history
- Informed consent
- Routine preoperative assessment such as Mallampatti grade, BMI, oral cavity examination.

*Perioperative management*

- Adequate analgesia in preoperative, intraoperative, infiltration at the end of surgery and postoperative period
- Adequate premedication to smoothen the anesthesia and postoperative recovery
- Avoidance of opioids to avoid postoperative delirium and vomiting
- Perform surgery with as minimal access as possible and care to achieve optimum hemostasis with minimal tissue handling and dissection
- Procedures such as varicose vein surgery, hemorrhoidectomy, inguinal hernia, TURP, hydrocele, varicocele, circumcision, tonsillectomy are commonly performed in day care fashion.

*Discharge*

Patient is to be discharged only if:

- He has no complaints of pain, nausea, vomiting
- He is accepting orally and able to pass urine
- He is conscious, oriented and vitally stable
- Has no surgical complications such as reactionary hemorrhage or dressing soakage and
- Has a good social support system as mentioned above.

**Advantages**

- Patient benefits because of the rapid recovery and early return to routine life with minimal physiological changes, less cost and less postoperative complications
- Hospital benefits because of the rapid patient turnover and economical use of resources.

**Q39. Write in brief about robotic surgery.**

**Ans.** Robotic surgery is telesurgery, i.e. the surgeon performs surgery by being away from patient through a console which manipulates the robotic instruments according to surgeon maneuvers on the console.

**The robotic system used now is DaVinci system.**

**Parts**

- **Master surgeon console:** On which the surgeon sits, uses his hands to manoeuvre instruments via master controllers and sees 3 -dimensions vision through the stereoviewer
- **Patient cart:** Patient lies on this cart and the instruments are docked (fixed) into the ports on the robot wheel cart over this table. There are 4 arms for instruments with one of them being camera port. Ports are placed as in laparoscopy and then the instruments inserted in ports through robot arms to connect them to the master console control
- **Vision cart:** Has a binocular vision through 3-D endoscope connected to camera.

**Advantages**

- Stereoscopic depth perception (3-D vision)
- More range of motion due to wrist like motion present in robotic instruments which give some degrees of freedom in movement
- Improved hand eye co-ordination as the robotic camera is controlled by the surgeon and is held in steady position by robotic cart so no fatigue or tremors
- Less surgeon fatigue due to excellent ergonomics (sitting surgery)
- Comparably less prolonged learning curves than with laparoscopy.

**Limitations**

- Cost
- Learning curve
- Duration of surgery is prolonged compared to open surgery

**Commonly performed robotic procedures**

- Gastrointestinal—cholecystectomy, pancreatic resections, bariatric surgery, colectomy, mesorectal excision, fundoplication, gastrectomy
- Urology—radical cystectomy, prostatectomy, pyeloplasty, nephrectomy
- Thoracoscopic—esophageal surgery and thymectomy
- Head and neck—transoral robotic surgery (TORS) for nasopharyngeal cancer, robotic thyroidectomy
- Gynecology—hysterectomy

## SURGICAL INFECTIONS

**Q40. Enumerate the causes of generalized lymphadenopathy. Discuss tubercular cervical lymphadenopathy.**

**Ans.**

**The causes of generalised lymphadenopathy are as follows:**

<b>Infections</b>	<ul style="list-style-type: none"> <li>• Tuberculosis, brucellosis, syphilis</li> <li>• HIV, infectious mononucleosis</li> <li>• Toxoplasmosis</li> </ul>
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*Contd...*

Contd...

<b>Malignancy</b>	<ul style="list-style-type: none"> <li>• Lymphoma</li> <li>• Leukemia</li> </ul>
<b>Autoimmune disorders</b>	<ul style="list-style-type: none"> <li>• SLE</li> <li>• Rheumatoid arthritis</li> <li>• Sarcoidoses</li> <li>• Amyloidoses</li> </ul>
<b>Other</b>	<ul style="list-style-type: none"> <li>• Metastasis from various malignancies.</li> </ul>

### **Tubercular cervical lymphadenopathy**

- It is a very common cause of cervical lymphadenopathy and should be ruled out in all cases of it
- Caused by *M. tuberculosis*
- Children are more commonly involved but, it can affect individuals from any age group.

### **Clinical features**

- Fever with evening rise, anorexia, weight loss, chronic cough with sputum production might precede the appearance of cervical lymph nodes
- Initially, the nodes are enlarged and discrete. This is due to lymphadenitis without periadenitis
- Then, lymph nodes become matted due to periadenitis and adhesions
- There is no sign of inflammation and the nodes are nontender
- Upper jugular cervical nodes are most commonly involved. Other groups and lymph nodes in other areas can also be involved.

### **Natural history**

- If the disease progresses unhindered, then after matting of lymph nodes, caseous necrosis leads to abscess formation which is known as **cold abscess**.
- Further progression lead to tracking of this pus along the fascial layers of neck. Because of the plane of lymph nodes deep to deep cervical fascia, the pus also travels down deep to this fascial layer
- Further increase in pus leads to increased tension and rupture of deep cervical fascia with tracking of pus in superficial fascia. This is known as **collar- stud abscess**. It shows cross-fluctuation due to presence of two abscess cavities on either side of the deep fascial rupture
- Finally, the pus ruptures through the superficial fascia and skin and presents as **tubercular sinus or ulcer**.

### **Investigations**

- Evaluation for tuberculosis—chest X-ray, sputum for AFB and culture, mantoux test, ESR, total leukocyte count with differential count, hemoglobin level.
- FNAC of the lymph node and if it is inconclusive, excision or incision biopsy can be done
- CECT neck and chest will show the entire extent in cases with cold abscess.

### **Treatment**

- Antitubercular medications as described in the question on ATT
- Local management of abscess is done by antigravity aspiration that is aspiration from higher level than the most dependent part if it is large or if it does not resolve on anti-tubercular treatment.

- Excision is done for enlarged lymphnodes if they do not resolve with antitubercular treatment.

**Q41. Discuss the medical management of a patient of TB.**

**Define the various types of drug resistant TB categories and discuss their management.**

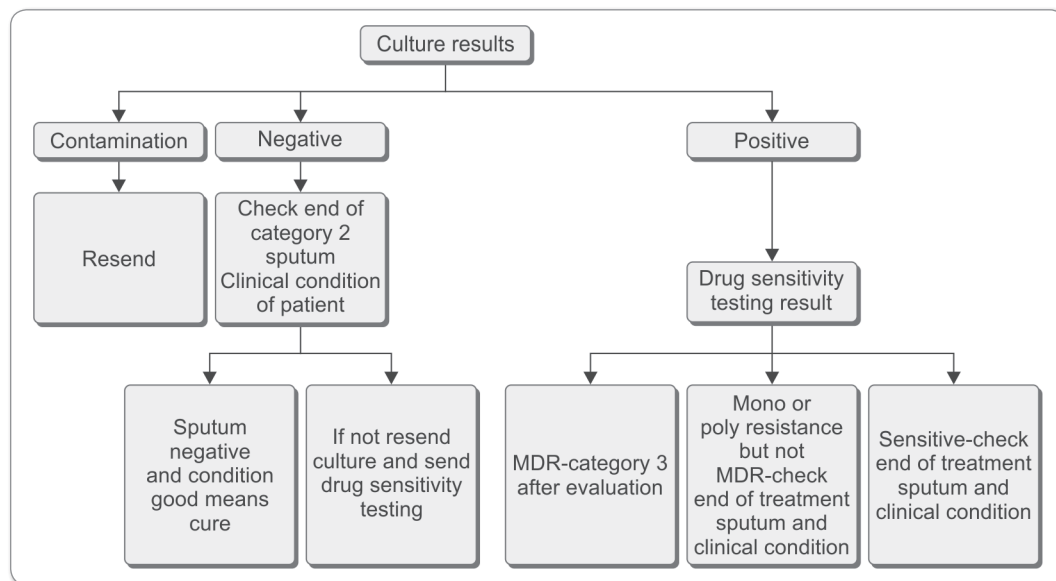
**Ans.**

- DOTS categories

<b>Category 1</b>	New case pulmonary or extrapulmonary	2HRZE+ 4HR
<b>Category 2</b>	Relapse, failure, default	2HRZES + 1 HRZE + 5HRE
<b>Category 3</b>	MDR TB	6 drug IP for 6–9 months 4 drug CP for 18 months

H – isoniazid, R – rifampicin, Z – pyrazinamide, E – ethambutol, S – streptomycin

- **MDR-TB suspect**
  - Any patient who fails category 1 treatment
  - Category 2 patients who remain sputum positive at 4 months of treatment or later
  - Close contacts of MDR TB patients with smear positive pulmonary TB
- MDR TB suspect patient—start appropriate ATT first according to class of the patient



**MDR TB case:** MDR TB suspect with sputum culture positive with resistance to atleast H and R. All patients with MDR TB and all patients with rifampicin resistance are treated with category 3.

**Management of MDR TB**

- Follow-up smear and culture of specimens collected at the end of 3, 4, 5, 6, 7 months, then at 9th month end, 12th, 15th, 18th, 21th, 24th month end.

- 2 consecutive smear and culture negative samples obtained then patient is called smear/culture converted.

### Regimen (category 3)

Intensive phase	Kanamycin Ofloxacin/levofloxacin Ethionamide Ethambutol Pyrazinamide Cycloserine	6–9 months
Continuation phase	Ofloxacin/levofloxacin Ethionamide Ethambutol Cycloserine	18 months

PAS can be substituted if any of the above drug is not tolerated.

### Groups of anti-TB drugs

1	Oral 1st line	HRZE
2	Injectable aminoglycoside	Streptomycin, kanamycin, capreomycin, amikacin, viomycin
3	Fluoroquinolones	Ciplox, oflox, levoflox, moxiflox, gatiflox
4	Oral 2nd line	PAS, ethionamide, cycloserine
5	Other drugs with unclear efficacy	Linezolid, amoxicillin, thiacetazone

### XDR TB

- Extensive drug resistant TB
- H, R resistance + any fluoroquinolone resistance + at least one of three injectable anti-TB resistance
- Treat with category 3 treatment as per sensitivity

### Totally drug-resistant TB (XXDR- TB/ virtually untreatable TB/ extremely drug-resistant TB)

- All first line oral + second line oral + all injectable
- These are also treated like XDR TB with maximum possible culture sensitive drugs for > 2 years.

### Q42. Write a note on Ludwig's angina.

Ans.

#### Definition

Rapidly spreading cellulitis of floor of mouth and submandibular space (submaxillary or submental and sublingual divided by mylohyoid) secondary to:

- Soft tissue infection
- Tonsil infection
- Lower premolar or molar infection (80%)
- Submandibular sialadenitis
- Injuries to the oral mucosa

**Symptoms**

- Trismus,odynophagia
- Excessive salivation
- Tongue is pushed upwards and backwards due to edema of the floor of the mouth.

**Extent**

- Can go to parapharyngeal and retropharyngeal spaces
- Laryngeal edema
- Aspiration pneumonia
- Septicemia

**On examination**

- Tongue is pushed backwards
- Woody hard feel of submandicular and submental spaces

**Organisms**

- Mixed infection of aerobes and anaerobes
- Dental origin—streptococcus viridians and E. Coli

**Treatment**

- Systemic antibiotics
- Tracheostomy if airway is endangered
- Incision and drainage should be delayed as long as possible, as pus is seldom found
- Submaxillary space (superficial to mylohyoid)—transcutaneous drainage through submandibular skin fold
- Sublingual space (deep to mylohyoid)—drained through floor of mouth

**Q43. Write a note on tetanus.**

**What is tetanus neonatorum? Discuss its preventive measures.**

**Write the measures to be taken for prophylaxis of tetanus after an injury.**

**Ans.**

- Tetanus is caused by clostridium tetani—a gram positive, anaerobic, terminal spore bearing organism
- It produces exotoxin called tetanospasmin which on reaching the neural tissue acts on spinal cord, brain, sympathetic system and motor end plate of muscles to block the normal inhibitory pathways of these nerves and lead to the characteristic muscular rigidity and spasms
- The spores reside in soil and dust and enters body through contaminated wound and injuries or chronic ulcers or dental extraction sites or in injection drug abusers through injection sites from where they germinate, produce exotoxin and bind their neuroreceptors to cause the disease. This entire life cycle takes around 5 to 10 days in the body
- Some specific modes of transfer give the tetanus its name.
  - **Tetanus neonatorum**—infection through infected cut surface of umbilical cord

- **Puerperal or postabortal tetanus**—infection through unsterile uterine instrumentation.
- **Otogenic tetanus**—infected material to clean ear can introduce infection
- Clinical features
  - **Most early symptom**—trismus (Lock jaw)
  - Anxious expression of face (Risus sardonicus or sardonic grin)
  - Opisthotonus
  - Constitutional symptoms such as elevated temperature, tachycardia, tachypnea, cyanosis
  - Rigidity of muscles of lower limb and abdomen. Gradually all the muscles develop rigidity and death is due to respiratory arrest, due to laryngeal spasm and spasm of respiratory muscles.
  - Other causes of death include aspiration pneumonia, lung infections or hyperpyrexia.

### Management of established case

- Patient should be admitted to a quiet, dark and well ventilated room
- Care of airway, breathing
- Sedation with diazepam/barbiturate is frequently used to avoid provocation of spasms
- Foley catheter, feeding tube, muscle relaxation and tracheostomy, IV fluids and antibiotics as per requirement. These are needed in severe cases. Mild cases can be managed with conservative measures and supportive care alone
- General nursing care of the patient till he stabilizes and is out of the ventilator.

### Specific treatment

- Passive immunisation with human anti-tetanus globulin, 4000 units or equine anti-tetanus serum, 1 lac units half IV and half IM should be administered with tetanus toxoid (active immunisation).
- Crystalline penicillin 20 lac unit 6 hourly or 10 lac unit 4 hourly should be given
- Wound debridement.

### Prophylaxis

**Means:** Immunization, antibiotic and wound cleansing and **debridement (most important).**

Active immunization	Passive immunization
<ul style="list-style-type: none"> <li>• Aluminium phosphate adsorbed tetanus toxoid 0.5 mL in left deltoid is the best agent for active immunization</li> <li>• 3 doses at 4 weeks interval and then booster after 1 year completes the routine mandatory immunization requirement. Booster is given at every 4–5 years to maintain immunity. Immunization schedule is 6,10,14 weeks age, 18 months age, 5 year age and 10 year age</li> </ul>	<ul style="list-style-type: none"> <li>• Equine anti-tetanus serum 1500 units SC/ IM after negative sensitivity testing</li> <li>• Human anti-tetanus globulin 250–500 units IM is a homologous antitoxin and does not require sensitivity testing. It is many times more protective than equine serum and is therefore preferred</li> </ul>

### Plan of immunization after injury

- A patient who has been completely immunized as above within past 5 years needs no tetanus immunization prophylaxis. It should be managed only with wound debridement



- A patient who has been completely immunized in past with a clean non-penetrating wound < 6 hours duration needs only a single tetanus toxoid injection and wound debridement
- A patient who has been completely immunized in 5–10 years range with a dirty, penetrating or old wound needs only a single tetanus toxoid injection and wound debridement but, if it is > 10 years then he needs single shot tetanus toxoid with anti-tetanus globulin.
- A patient whose immunization history is not known needs complete tetanus immunization course if he has a clean nonpenetrating wound < 6 hours duration and complete tetanus immunization course with anti-tetanus globulin for old, penetrating or dirty wounds.
- **Tetanus neonatorum** can be prevented by using aseptic techniques and surroundings during delivery and aseptic means to cut the cord. Also 2 maternal immunizations at 16 and 20 weeks serve to prevent both maternal and neonatal tetanus in nonimmunized mothers and a single booster dose in immunized mothers. The immunization can be given any time the mother is seen but atleast three weeks before delivery. If the mother is seen directly during delivery, then the infant can be given 750 units Equine ATS or 250 units human serum within 6 hours of birth to prevent tetanus neonatorum.

**Q44. Write a note on gas gangrene.**

**What is gas gangrene? Discuss its management.**

**Ans.** Gas gangrene is caused by clostridium perfringens/clostridium welchii and other clostridia species such as *C. septicum*, *C. histolyticum*, etc., anaerobic organisms also called “flesh eating bacteria”.

**Pathogenesis**

- These organisms enter the body through wounds which are grossly contaminated with soil/foreign bodies which provide the low tissue resistance, low pH, anaerobic environment and high calcium due to cell lysis in the wound necessary for clostridial growth
- The organisms are also present in stools and female genital organs and therefore wounds that come in contact with these areas and body fluids are more predisposed
- The organism then produce exotoxins which cause the characteristic gas gangrene. These include lecithinase (Alpha toxin), hyaluronidase, collagenase, other hemolytic and leukocidal toxins
- Occlusion of end arteries supplying the wound, accumulation of wound exudates and inadequate wound debridement are all factors that increase the risk of occurrence of gas gangrene
- Diabetes and chronic arterial occlusive diseases also predispose.

**Stages of disease progression**

1. Stage of contamination—no symptoms and signs.
2. Stage of anaerobic clostridial cellulitis—no systemic symptoms.
3. Spreading cellulitis and fascitis with systemic symptoms and signs.
4. Clostridial myonecrosis.
5. Bacteremia and septicemia.

**Clinical features**

- It presents as rapidly spreading infective gangrene with accumulation of gas in muscles and subcutaneous tissue and is called clostridial myonecrosis
- Initially it can be present as clostridial cellulitis with edema of subcutaneous tissue and spreads rapidly to involve the muscles and subcutaneous tissue rapidly
- Can affect the liver (Foaming liver)
- Constitutional symptoms such as pyrexia, anxiety, tachycardia, vomiting and hypothermia are initial symptoms
- Pain out of proportion to the appearance of wound, swelling and edema of the affected part, profuse, foul smelling, brown or grey color discharge called “dishwater pus” are also the characteristic features. Finally, the wound becomes black in color marking the phase of gas gangrene
- On examination, crepitus is almost always present and is pathognomonic.

**Investigations**

- Gram stain of wound exudates reveal the organisms
- Anaerobic cultures are positive
- Nagler’s reaction is used to detect *C. welchii*.

**Management***Treatment of a diagnosed case*

- Wound drainage and debridement can extend from extensive debridement to even guillotine type amputation as a life saving measure in cases of severe myonecrosis
- Crystalline penicillin 10 lac unit 4 hourly or 20 lac unit 6 hourly
- Anti-gas gangrene serum 22,500 IU (contains 9000 units *C. welchii*, 4500 units *C. septicum* and 9000 units *Cl. oedematiens*) stat and repeated at 6-hour interval for 3 consecutive doses
- Hyperbaric oxygen therapy is also used once the wound is non-infective and healthy to aid healing.

*Prophylaxis*

- Wound drainage and debridement to remove all devitalized tissues and foreign bodies
- Crystalline penicillin 10 lac unit 4hrly or 20 lac unit 6hourly
- Anti gas gangrene serum 22,500 IU (contains 9000 units *C. Welchii*, 4500 units *C. septicum* and 9000 units *Cl. oedematiens*) stat single dose is suggested but not of proven role in prophylaxis.

**Q45. What is carbuncle? Discuss its management.**

**Ans.** Carbuncle is caused by staphylococcal infection (*S. aureus*)—aerobic, gram positive cocci in clusters and is an infection of subcutaneous tissue.

**Pathogenesis**

- The organism penetrates deep into subcutaneous tissue and produces multiple interlinked abscesses that open separately on surface to produce a sieve like appearance
- They coalesce in center and produce the wound
- This can extend peripherally and assume large sizes

**Clinical features**

- Patients are commonly diabetic males
- Common sites are nape of neck, back, dorsum of hand, shoulder, chest and abdomen
- Constitutional symptoms mark the onset
- Begins as a painful, edematous subcutaneous swelling and progresses to a sieve like appearance which is characteristic of carbuncle
- The openings can finally enlarge, coalesce and form an ulcer.

**Investigations**

- Wound smears and culture sensitivity
- Blood culture
- Diabetic work up

**Treatment**

- Control of diabetes
- Wound debridement and dressing—cruciate incision and derroofing
- Culture specific antibiotic therapy

**Q46. Write a note on madura foot.****Ans.**

- Madura foot is an infectious condition affecting foot
- It is also known as mycetoma.

**Causes are as follows:**

Eumycetoma	Actinomycetoma
<ul style="list-style-type: none"> <li>• Organism—<i>Madurella mycetomeii</i>.</li> <li>• Disease is slowly progressive type</li> <li>• Treatment is antifungal therapy for prolonged periods</li> </ul>	<ul style="list-style-type: none"> <li>• Organism—<i>Actinomyces</i>, <i>Nocardia</i></li> <li>• Disease is rapidly spreading and extensive.</li> <li>• Treatment is amikacin + co-trimoxazole (first line) or rifampicin or amoxycylav (second line)</li> </ul>

**Entry and route of spread**

- Inoculation from trauma site
- From here the disease localizes in the site and further spread is via lymphatics as well as along the fascial planes

**Clinical features**

- **Triad** of noncontagious multiple sinuses, multiple painless subcutaneous masses and seropurulent discharge
- **Site:** Foot is the disease site in most cases. Other sites are perineum, arm, leg, head, neck, thigh and hand
- Tendons and nerves are spared till very late in the disease
- Secondary bacterial infection can complicate the local disease
- The disease spreads along the fascial planes so what is a small lesion superficially might be extending for large areas deeply.

**Diagnoses**

- **X-ray**—areas of calcification can be seen. Also, areas of punched out lesions in bone can be seen.
- **Ultrasound**
- **MRI**—shows “dot in circle” sign.
- **Biopsy:**
  - **Type 1 mycetoma:** Multiple neutrophils surrounded by lymphocytes and mononuclear cells surrounded by fibroses.
  - **Type 2 mycetoma:** There are no neutrophils in the lesion. It only contains macrophages.
  - **Type 3 mycetoma:** It contains epithelioid granulomas with giant cells.
- **FNAC** can also be done but biopsy is more reliable.
- **Culture-sensitivity** is done to help in selection of culture specific antibiotics.

**Treatment**

- Antibiotics or antifungals as written above
- Surgical debridement under general or spinal regional anesthesia. Use of local anesthesia is contraindicated.

**Q47. Write a note on anatomy of palmar spaces.**

**Ans.**

- Arrangement of the fascia and fascial septa in the hand is such that it forms many spaces
- Spaces are of surgical importance because they may become infected and distended with pus
- The three palmar septa divide the palmar space into thenar and midpalmar spaces.

**Midpalmar and thenar spaces**

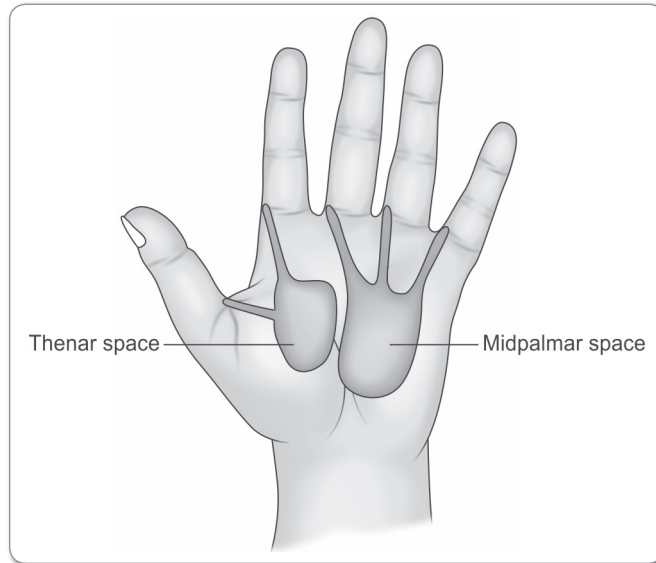
- These are triangular spaces of which base lies distally and the apex is directed proximally
- The thenar space lies between the lateral and intermediate palmar septa
- The midpalmar space lies between the intermediate and medial palmar septa.

**Boundaries of thenar space**

- Medially: Intermediate palmar septum
- Laterally: Lateral palmar septum
- Anteriorly: Lateral part of palmar aponeurosis, and flexor tendons to index finger
- Posteriorly: Adductor pollicis, transverse head.

**Boundaries of midpalmar space**

- Medially: Medial palmar septum
- Laterally: Intermediate palmar septum
- Anteriorly: Medial part of palmar aponeurosis and flexor tendons to medial three fingers
- Posteriorly: Fascia covering the medial three metacarpal bones and intervening interosseous muscles.



**Fig. 1:** Anatomy of palmar spaces

#### **Extent of spaces**

- Proximally, the midpalmar and thenar spaces extend up to the distal margin of the flexor retinaculum
- Distally, the thenar space extends up to the proximal transverse crease of the palm and the midpalmar space extends up to the distal transverse crease
- Incisions are made through these creases to drain the spaces.

#### **Communications of spaces**

- The spaces are normally closed at the proximal end
- However, occasionally the midpalmar space may communicate with the forearm space through the carpal tunnel
- The two spaces of the palm quite frequently communicate with each other, and infection can pass from one to the other.

#### **Contents of the thenar and midpalmar spaces**

- Normally filled mainly with loose connective tissue. When infected they can be distended with pus
- They are closely related to the lumbrical muscles. The thenar space contains the first lumbrical muscle, while the midpalmar space contains the second, third and fourth lumbrical muscles, the tendon of each lumbrical muscle is surrounded by a lumbrical canal.

#### **Clinical Correlations**

- Infection of the midpalmar space may result from tenosynovitis of the middle and ring fingers or from a web infection which has spread proximally through the lumbrical canals
- When this happens the normal concavity of the palm is obliterated and the swelling extends to the dorsum of the hand

- The space can be drained by an incision in either the 3rd or 4th web depending on where the pus points.

**Q48. What is the difference between acute paronychia and felon? Discuss its features.**

**Write a note on acute paronychia.**

**Ans. Definition**

- Acute paronychium is the infection of the nail fold whereas felon or whitlow is terminal pulp space infection
- Acute paronychium is the most common hand infection whereas felon is the second most common infection.

**Etiology**

- Small pricks and abrasions and cuts in hand especially in people involved with household work, manual laborers, gardeners is the commonest etiological factor
- Unsterile manicure instruments can also result in paronychium
- *S. aureus* is the most common organism.

**Clinical features**

*Paronychia*

- Painful swollen nail folds is the most common presentation
- Examination reveals tenderness, redness and swelling of the nail fold which increases on pressing the nail
- Suppuration can extend deep to nail and reach upto base of nail.

*Felon*

- The pulp of finger is painful and swollen
- Examination reveals tenderness, redness and swelling of pulp which increases on pressing.

**Complications**

*Acute paronychium*

- Chronic paronychium
- Paronychium can extend into pulp space and result in felon.

*Felon*

- Suppurative flexor tenosynovitis
- Septic arthritis of distal interphalangeal joint
- Osteomyelitis of terminal phalanx.

**Treatment**

*Acute paronychium*

- Conservative management with antibiotics and analgesics in mild cases
- Elevation of nail fold and release of pus
- Bilateral cuticular incision and pus drainage
- Nail excision and pus drainage

*Felon*

- Conservative management with antibiotics and analgesics in mild cases
- Incision at the pus point and drainage of pus
- Lateral pulp incision and drainage of pus
- Management of complications if they are present

**Q49. Write a note on life cycle of hydatid cyst and the diseases caused by it.**

**Ans.** Hydatid cyst is caused by *Echinococcus* genus which has following species

- *E. granulosus* (Dog tapeworm or hydatid worm)—hydatid cyst
- *E. oligarthus* and *E. vogeli*—polycystic hydatid disease
- *E. multilocularis*—alveolar or multilocular disease

**Definitive hosts:** Dogs, wolves and jackal harbor adult worms and eggs of the parasite.

**Intermediate hosts:** Man, cattle and sheep harbor the larval form of the parasite.

**Transmission**

- Dogs are infected by eating contaminated meat of intermediate hosts
- Intermediate hosts are infected through ingestion of eggs passed in dog feces through contaminated water or food or through dog handling.

**Life cycle**

- The adult worms live in the small intestine of definitive hosts and discharge eggs in their feces
- The larvae hatch after 6–8 hours in the small intestine of the intermediate hosts, penetrate the intestinal wall and reach liver through portal venules
- In liver, some get filtered and stay in liver while some larvae escape and reach lungs
- Again some get filtered while others reach the systemic circulation and reach the other organs such as brain, kidney, bone, spleen or eye where it matures and forms the hydatid cyst.

**Disease**

- **Liver hydatid (discussed in liver chapter)**
- **Pulmonary hydatid**—can present as cough, hemoptysis, empyema or rupture and pneumothorax, anaphylaxis, hemoptysis.
- **Cerebral hydatid** can be present with new onset seizures.
- **Can also be present in spleen, bones, eye and heart.**

**Q50. Write a note on life cycle of *Entamoeba histolytica* and diseases caused by it.**

**Ans.**

**Life cycle**

- The *E. histolytica* passes its life cycle in single host
- The *E. histolytica* has two forms in life—the trophozoite and the cyst
- The infective form is the mature cyst in feces of carriers and reaches the human host by fecal or oral transmission
- The cyst wall is damaged by trypsin in lower ileum or cecum and this leads to excystation and liberation of a single ameba—the quadrinucleate trophozoite

- This trophozoite divides its contents to form 8 small metacystic trophozoites each of which mature into a trophozoite
- The trophozoites thus formed lodge in the submucous glandular crypts in colon and form cysts which are passed in feces and the cycle continues.

### Diseases

- **Amebic colitis and amebic ulcers**
  - These occur because of penetration of the amebic trophozoites into the colonic mucosa and submucosa with help of enzymes such as histiolysin and formation of transverse ulcers with ragged undermined edges and base on the muscular coat
  - The trophozoites enter the portal radicles and are carried to extraintestinal sites
  - Can result in **rectovesical fistula or rectocutaneous fistula**.
- **Amebic liver abscess** (Discussed in liver section)
- **Ameboma**: Inadequately treated amebic colitis result in formation of a chronic amebic mass in the cecum which is called ameboma. It is a tumor like lesion and consists of granulomas. It is commonly confused with malignancy and needs differentiation before labeling it as ameboma.
- **Pulmonary amebiasis**
  - **Primary pulmonary amebiasis** occurs when the ameba reaches lungs through the pulmonary circulation
  - **Secondary pulmonary amebiasis** occurs due to direct extension of liver abscess through the diaphragm into lungs.
- **Splenic amebiasis**
- **Cerebral amebiasis**
- **Amebiasis cutis**—cutaneous amebic abscess.

### Q51. Write a note on actinomycosis

Ans.

#### Etiology

- *Actinomyces Israelii* is an anaerobic gram-positive fungus like bacterium and a branching filamentous organism (Ray fungus)
- Commonly found as normal flora of the oral cavity (within gingival crevices, tonsillar crypts, periodontal pockets, dental plaques, and carious teeth), pharynx, tracheobronchial tree, gastrointestinal tract and female urogenital tract

#### Pathogenesis

- Organism enters through deeper plane of the tissue causes subacute inflammation with induration and nodule formation. Eventually discharging sinus forms at the surface. Pus collected in a swab or sterile tube will show sulphur granules.

#### Clinical presentation

- **Cervicofacial disease** (most common site):
  - Occurs in the setting of poor dental hygiene, recent dental surgery or minor oral trauma
  - Painful soft tissue swelling commonly seen at the angle of the mandible
  - Fever, chills, and weight loss



- Trismus
- Soft tissue facial infection with sinus tract or fistula formation.
- **Thoracic disease:**
  - Can involve the lungs, pleura, mediastinum, or chest wall
  - Presumed secondary to aspiration of *Actinomyces* organisms in patients with poor oral hygiene
  - Fever, cough, weight loss, and pleuritic chest pains are common symptoms
  - Signs of pneumonia or pleural effusion may be present
  - With extension beyond the lungs to mediastinal structures and the chest wall, signs and symptoms of pericarditis, empyema, chest wall sinus drainage and
  - Tracheoesophageal fistula can also occur.
- **Abdominal disease:**
  - Occurs most commonly after appendectomy, perforated bowel, diverticulitis or surgery to the gastrointestinal tract.
  - Lesions develop most commonly in the ileocecal valve, causing abdominal pain, fever, weight loss, and a palpable mass.
  - Extension may occur to the liver, causing jaundice and abscess formation.
  - Sinus tracts to the abdominal wall can occur.
- **Pelvic disease:**
  - Commonly occurs by extension from abdominal disease of the ileocecal valve to the right adnexa (80% of cases).
  - Endometritis.

### Differential Diagnosis

- Cervicofacial disease: Odontogenic abscesses, brachial cleft cyst
- Pulmonary disease: Tuberculosis, nocardiosis, botryomycosis, chromomycosis, fungal disease of the lung
- Intestinal disease: Intestinal tuberculosis, ameboma, colon cancer, inflammatory bowel disease
- Pelvic disease: Pelvic inflammatory disease, Crohn's disease
- CNS disease: Brain abscess, brain tumors, toxoplasmosis, intracranial hematoma.

### Investigation

- Isolating "sulfur granules" from tissue specimens or draining sinuses confirms the diagnosis of actinomycosis.
- Sulfur granules are nests of actinomyces species. Sulfur granules may be macroscopic or microscopic. Sulfur granules are crushed and stained for identification of actinomyces organisms and may take up to 3 weeks to grow in culture media.

### Treatment

#### *Nonpharmacologic therapy*

- Incision and drainage of abscesses
- Excision of sinus tract.

#### *Treatment for acute phase*

- Penicillin 10 to 20 lac units per day in 4 divided doses for 4 to 6 weeks

- In penicillin-allergic patients, erythromycin, tetracycline, clindamycin or cephalosporins are reasonable alternatives
- Chloramphenicol 50 to 60 mg/kg/day can be used for CNS actinomycosis.

#### *Treatment for chronic phase*

- Following 4 to 6 weeks IV penicillin, oral penicillin V 500 mg PO 4 times a day for 6 to 12 months
- Longer period of treatment is needed to eradicate the disease.

## TRAUMA AND DAMAGE CONTROL SURGERY

### **Q52. Write a note on flail chest.**

**Ans.** Flail chest occurs when two or more consecutive ribs fracture at two or more sites each. This results in a segment of rib cage that is not in continuity with the rest.

#### **Pathophysiology**

This segment moves opposite to the rest of the chest, i.e. it moves inwards during inspiration and outwards during expiration. This is called paradoxical respiration which is the characteristic sign of flail chest. This causes two main problems:

- It causes mechanical injury and lung contusions which leads to impaired lung function in that area
- The paradoxical movement affects ventilation and causes accumulation of carbon dioxide in that area and hypoxia.

#### **Other effects**

- Paradoxical respiration leads to mediastinal shifts which can lead to shock due to its effects on heart and great vessels
- Decreased respiratory excursion due to pain also leads to ventilation defects
- Accumulation of bronchopulmonary secretions also adds to the pulmonary insult.

#### **Anatomical types**

- **Anterior type:** Ribs get detached/fractured from both sides of sternum so that the part of the sternum becomes the flail segment
- **Lateral type:** The ribs are fractured anteriorly and posteriorly so that lateral aspect acts as the flail segment
- **Posterior type:** Ribs are fractured on either side of angle of rib posteriorly so that the spinal segment becomes the floating segment.

#### **Clinical features**

- Breathlessness
- Chest pain which increases on deep breathing, coughing, speaking
- Shock

#### **Treatment**

- Pain relief with adequate analgesia

- Respiratory support as per necessity can be oxygen by mask/ noninvasive or invasive positive pressure ventilation (IPPV) using endotracheal tube or tracheostomy
- External stainless steel wire fixation or open operative K- wire fixation is to be considered in severe cases of respiratory compromise.

**Q53. Write a note on field management of disaster.**

**Ans.**

**Definition of disaster**

A disaster is a sudden, calamitous event that seriously disrupts the functioning of a community or society and causes human, material and economic or environmental losses that exceed the community's or society's ability to cope using its own resources. Though often caused by nature, disasters can have human origins.

**A disaster occurs when a hazard impacts on vulnerable people**

The combination of hazards, vulnerability and inability to reduce the potential negative consequences of risk results in disaster

In medical terms, any loss of life is a disaster. However, when a true disaster strikes, all resources need to be utilized in order to appropriately respond to it.

**Problems in a disaster**

- Communication
- Access to the affected area
- Time frame of disaster
- State of development of the area and nation.

**Common management**

- Appoint a leader in charge of disaster management
- Mobilize disaster team
- Establish communication
- Start rescue operations
- Provision of shelter food and water with medical facilities is very important
- Media law and order
- Role of medics and paramedics.

**Triage casualties**

- Red—immediate treatment
- Yellow—urgent treatment
- Green—non-urgent
- Black—unsalvageable.

Triage helps to provide greater goods to many patients/people and at the same time decreases load on medical services.

Triage is to be done at all levels of health care. Patient is shifted according to triage to health care setups.

*Triage is discussed as a separate short note*

Prevent "second accident" while shifting

**Field management of disaster**

Field hospitals can be “tent type” or “modular type.”

*Field hospital management includes*

- First aid
- Damage control surgery of field hospitals
- Management of non-life-threatening injuries

**Damage control is discussed in the next question.**

**Q54. What do you mean by “damage control” in surgery?**

**What is damage control surgery in moribund patient?**

**Ans.**

A. Damage control resuscitation
B Damage control surgery in field hospital
C Damage control surgery of moribund

When asked as damage control surgery it is better to write both 2 and 3 in answer.

**A. Damage control resuscitation**

*Basis:*

- Crystalloids cause neutrophil activation and increased inflammation
- This approach decreases ARDS by 25 % to 9 % in ICU
- D (–) lactate causes psychoneurotic disturbances than L(+) lactate

*Principles :*

- Permissive hypotension till control of bleeding with or without damage control surgery
- Limit crystalloid and colloid use as it can cause dilutional coagulopathy
- Anticipate and treat acute traumatic coagulopathy with transfusion of FFP, platelets cryoprecipitates, factor 7a and factor 9, hypertonic saline, PRBCs
- Avoid hypothermia

*Principle is to transfuse 1:1:1 platelet : plasma : PRBCs*

- If ongoing bleeding and anticipated need of massive transfusion in patient (> or = 10 units PRBCs in 24 hours), then activate massive transfusion protocol
- This protocol allows transfusion of 2 units of uncrossmatched blood till availability of crossmatched blood and maintain a ratio of 1:1:1
- Platelet transfusion at 1 apheresis unit (or 6 RDP units) for 6 units of PRBCs to keep platelet count >1 lac/cu mm during acute hemorrhage control
- FFPs also given as 6 units for every 6 units PRBCs
- If fibrinogen is less than 100 mg/dL, give 20 units of cryoprecipitate and repeat as required
- Largest benefit in mortality when FFP and platelets are administered during first 6 hours of admission.

*Benefits of permissive hypotension*

- No bursting of clots

- Decrease crystalloid requirement
- Minimize use of acellular fluids.

### B. Damage control surgery in field hospital

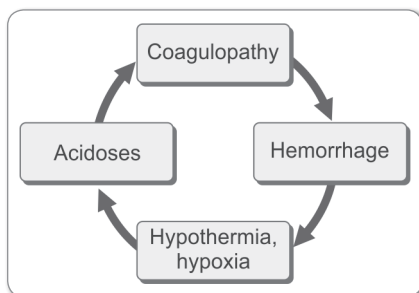
Damage control surgery in field hospital is different from damage control surgery in a moribund patient.

#### Principles

- To do minimum required management of patient so as to shift him to definitive facility
- Secure airway by ET tube or tracheostomy
- Hemorrhage control by pressure, laparotomy, craniotomy, thoracotomy, or repair of major limb vessels
- Prevent pressure build up in close compartments by burr hole, chest drain, laparotomy, fasciotomy
- Prevent infections by management of wounds by exposure or debridement.

### C. Damage control surgery in moribund patient

The aim of damage control in moribund that is physiologically exhausted patient is to stop patient from entering into the lethal triad of acidoses, coagulopathy and hypothermia.



These are the patients who are near or into the lethal triad and therefore prolonged surgery will not be tolerated by them.

Patient selection criteria and 5 phases of damage control surgery are given below:

<b>Physiological factors</b>	<ul style="list-style-type: none"> <li>• Hypothermia</li> <li>• Profound hypotension</li> <li>• Acidoses</li> <li>• Coagulopathy</li> <li>• Prolonged surgery needed for definitive repair &gt; 90 minutes</li> </ul>
<b>Complex injuries</b>	<ul style="list-style-type: none"> <li>• Multiple trauma injuries = penetrating or blunt</li> <li>• Combined vascular and visceral injuries</li> <li>• Multiple body cavities injury with competing management issues</li> </ul>
<b>Other considerations</b>	<ul style="list-style-type: none"> <li>• Planned resurgery</li> <li>• Injuries to be managed by nonoperative measures (embolisation, etc.)</li> <li>• Variable physiology (adults, athletes, females)</li> </ul>

- **Phase 1:** Involves control of hemorrhage and contamination. In this phase vessel injuries are just to be packed and not repaired (difference from damage control in field hospital)
- **Phase 2:** Involves resuscitation in ICU
- **Phase 3:** Involves definitive surgery with points as shown below
  - Inspection/identification of all the injuries
  - Control of all bleeding points
  - Careful removal of all previous packs
  - Definitive gastrointestinal and vascular repairs
  - Thorough abdominal wash
  - Radiography to rule out retained packs
  - Drains to be inserted if required
  - Feeding tubes if required
  - Stomas and tube enterostomies should now be avoided
  - Consideration for temporary or permanent abdominal wall closure.
- **Phase 4:** Involves planned ventral hernia if closure causes peak airway pressure to rise  $>10$  cm H<sub>2</sub>O
- **Phase 5:** Involves abdominal closure.

**Q55. Classify faciomaxillary trauma and write a note on Le Fort fractures.**

**Write a note on mandible fractures.**

**Write a note on maxillary fractures.**

**Ans. Faciomaxillary trauma include the following**

1. Orbit fracture	<ul style="list-style-type: none"> <li>• <b>Superior orbital fissure syndrome</b>—3,4,6 cranial nerves involved</li> <li>• <b>Orbital apex syndrome</b>—2,3,4,6 cranial nerves involved</li> <li>• <b>Most common site</b>—Floor &gt; medial wall</li> <li>• <b>Indications of surgery</b> include enophthalmos &gt; 2mm, fracture of &gt;50% of orbital floor, extraocular muscle entrapment, diplopia on primary or inferior gaze</li> <li>• <b>Access</b>—transconjunctival/subciliary or lower blepharoplasty incision</li> </ul>
2. Zygomatico-maxillary complex fracture	<ul style="list-style-type: none"> <li>• Zygomatic arch, lateral orbital wall, zygomaticofrontal and zygomaticomaxillary involved</li> <li>• Access—coronal incision or upper eyelid incision for zygomaticofrontal and lateral orbital</li> <li>• Access for orbital floor—tarsal/tarsoconjunctival incision</li> <li>• Access for zygomaticomaxillary—maxillary gingivobuccal sulcus incision</li> </ul>
3. Naso-orbito-ethmoid fracture	Plating or wiring all bones with or without primary bone grafting to be done
4. Frontal sinus fracture	<ul style="list-style-type: none"> <li>• Only anterior table involved and displaced—ORIF</li> <li>• Posterior table involved/CSF leak—ORIF for anterior table and for posterior table, remove posterior table bone, burr mucosa, obliterate Nasolacrimal duct, primary bone graft for posterior table and flap coverage of cavity</li> </ul>
5. Nose fracture	<ul style="list-style-type: none"> <li>• Incise septal hematoma and give antibiotics</li> <li>• Closed reduction</li> <li>• Reconstruction—nasolabial flaps and /or composite skin grafts can be used</li> </ul>

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6. Ear defects	<ul style="list-style-type: none"> <li>Small helical defects can be closed primarily</li> <li>Large defects—Antia Buch chondrocutaneous advancement flap</li> </ul>
7. Lip defects and reconstruction	<ul style="list-style-type: none"> <li><b>Upper lip</b> <ul style="list-style-type: none"> <li>&lt; 1/3 defect—primary closure</li> <li>1/3 to 2/3 defect—abbe flap (noncommisural) estlander flap (commisural), reverse karapandzic flap (midline), perialar crescentic advancement flap</li> <li>&gt; 2/3 defect—Burrow-Dieffenbach flap</li> </ul> </li> <li><b>Lower lip</b> <ul style="list-style-type: none"> <li>&lt; 1/3 defect—primary closure</li> <li>1/3 to 2/3 defect—abbe flap (noncommisural) estlander flap (commisural), karapandzic flap (midline)</li> <li>&gt;2/3 defect—Gillies fan flap, Webster-Bernard repair, radial forearm free flap.</li> </ul> </li> </ul>
8. Eyelid reconstruction	<ul style="list-style-type: none"> <li><b>Upper eyelid</b> <ul style="list-style-type: none"> <li>&lt;25% defect—primary closure</li> <li>25-50%—lateral canthotomy and cantholysis with advancement flap</li> <li>&gt;50%—cutler beard full thickness flap or modified Hughes tarsoconjunctival flap</li> </ul> </li> <li><b>Lower eyelid</b> Similar to upper eyelid by primary closure, lateral release and advancement, partial thickness or full thickness grafts for anterior lamella Fasanella-Servat procedure for ptosis</li> </ul>
9. Maxilla (Le fort) fracture	<p>Given by Le fort in 1911 which he described by looking at the pattern of skulls that he threw on ground from the terrace of a building</p> <ul style="list-style-type: none"> <li><b>Type 1</b>—seperates alveolus from the rest of the facial skeleton Fracture line runs from inferior pterygoid plates, nasal pyriform aperture and maxillary sinus</li> <li><b>Type 2</b>—also called pyramidal fracture Fracture line runs through middle of pterygoid plates and maxillary antrum, orbit, bridge of nose, ethmoids, with or without cribriform plate and infraorbital foramen</li> <li><b>Type 3</b>—separates the facial skeleton from the base of the skull Fracture line runs through nasal bridge, septum, ethmoids, orbit, fronto-maxillary suture and high through maxillary sinus and pterygoid plates.</li> </ul> <p><b>Approach</b></p> <ul style="list-style-type: none"> <li><b>Ideal time for surgery</b>—within 7 to 10 days after the original injury</li> <li><b>M.C. indication for early intervention</b>—restoration of the functional integrity</li> <li><b>Incisions</b> <ul style="list-style-type: none"> <li>Bicoronal—nasal root, frontozygomatic, orbital rim</li> <li>Lower eyelid blepharoplasty incision—orbital blowout fracture, infraorbital rim fracture treated with bone grafts, titanium mesh or alloplasts</li> <li>Gingivobuccal maxillary sulcus incision—maxillary fractures</li> </ul> </li> <li><b>Preserve dental occlusion</b> with the help of arch bars/Intermaxillary fixation screws/eyelet wires/maxillofacial fixation system with titanium fixtures. This system do not require 6 weeks immobilization</li> </ul> <p><b>Maxilla reconstruction</b></p> <ul style="list-style-type: none"> <li>Principles <ul style="list-style-type: none"> <li>Oronasal closure</li> </ul> </li> </ul>

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	<ul style="list-style-type: none"> <li>– Velopharyngeal competence</li> <li>– Dental rehabilitation</li> <li>– Height of cheeks</li> <li>– Height of eye globes</li> </ul> <ul style="list-style-type: none"> <li>• <b>Small defects</b> can be closed with obturators (malleable materials)</li> <li>• <b>Large defects</b> require flap reconstruction using deep circumflex iliac artery flap or vascularised muscle (rectus/latissimus) with nonvascularised bone graft</li> <li>• <b>Skin grafts with nasopharyngeal tube</b> used for nasal passages to reconstruct and at the same time prevent blockage of the airways.</li> </ul>
10. Mandible fracture and reconstruction	<p><b>Most common site</b>—neck of condyle fracture or defect can be <b>marginal</b> (continuity of angle to symphysis is intact) or <b>segmental</b> (continuity is broken)</p> <p><b>Anterior defect</b> is defect anterior to mental foramen whereas <b>lateral defect</b> is defect lateral to mental foramen.</p> <p>When segmental anterior deformity is not reconstructed it results in <b>andy gump deformity</b> and when lateral segmental defect is not reconstructed it causes dental malocclusion</p> <p><b>HCL classification of mandibular defects (Urken et al)</b> H—lateral defects of any length including condyle C—as above but condyle not included L—central segment (canine to canine)</p> <p><b>Angle classification of malocclusion</b> 1—normal alignment between mesial first maxillary molar and mandibular first molar 2—anterior displacement of maxillary first molar 3—posterior displacement of maxillary first molar</p> <p><b>Guardsman fracture:</b> Fracture of symphysis or parasymphysis on a direct blow to the chin point with unilateral or bilateral condylar fracture due to indirect energy transfer</p> <p><b>Indications of ORIF:</b> All bicondylar fracture or unicondylar displaced fracture</p> <p><b>Technique of mandibular plating:</b> AO/ASIF—rigid fixation or Champy-less rigid and functionally stable fixation.</p> <p><b>Options in reconstruction of defects</b></p> <ul style="list-style-type: none"> <li>• No bony reconstruction—soft tissue closure or interposition / alloplastic material (titanium or stainless steel)/local flaps</li> <li>• Bony reconstruction—vascularized muscle with nonvascularized bone/costocondral graft for TM joint/free bone graft</li> <li>• Vascularised bone—pedicled or free (fibula/scapula/DCIA)</li> <li>• Distraction histiogenesis (phases of osteotomies f/b latency f/b distraction at 1 mm/day f/b consolidation) can cover defects up to 6.5 cm</li> <li>• Sequence of reconstruction includes distraction histiogenesis f/b TMJ reconstructions f/b dental implants</li> </ul>
11. Cheek reconstruction of trauma defects	<ul style="list-style-type: none"> <li>• Pectoralis major myocutaneous flap (acromiothoracic artery)</li> <li>• Deltopectoral flap (perforators of internal mammary artery)</li> <li>• Radial artery forearm free flap</li> <li>• Temporoparietal forehead flap (anterior branch of superficial temporal artery)</li> </ul>



## PERIOPERATIVE SURGERY

### Q56. Write a note on intraoperative patient monitoring.

**Ans.** Intraoperative patient monitoring requires integration of clinical monitoring and electronic monitoring. Either alone would lead to increased chances of complications intraoperatively as well as postoperatively.

**American society of anaesthesiologists (ASA) recommends** the following standards of patient's intraoperative monitoring

<b>Standard 1</b>	A qualified anesthetist should be physically present in operation theater throughout the procedure.
<b>Standard 2</b>	He should actively monitor ventilation, oxygenation, temperature, circulation atleast and take appropriate corrective measures to keep them normal.

#### **Important monitors in operation theater include the following:**

- Temperature—esophageal, nasopharyngeal, pulmonary artery, tympanic membrane
- ECG—lead V4 is the most important lead to monitor for perioperative MI. Lead 2, V2, V4 are minimum leads to have to monitor a patient
- Alarms to detect low oxygen supply and system disconnection
- Pulse oximetry
- EtCO<sub>2</sub>—normal EtCO<sub>2</sub> and PaCO<sub>2</sub> differ by 2–5 mm Hg. This helps in detection of embolism/cardiac events
- Blood pressure
  - Noninvasive or
  - Invasive—radial A, ulnar A, axillary A, brachial A, femoral A, posterior tibial A or dorsalis pedis artery
- Blood glucose levels
- Urine output and fluid intake
- Monitoring of comorbidities and appropriate corrective measures
- **Neuromuscular blockade evaluation**
  - Train of four fade phenomenon—4 stimuli of 200 microseconds each given over 2 seconds period and then look for twitch
  - If 4th stimuli produce no twitch—75% blockade
  - If 3rd stimuli produce no twitch—80% blockade
  - If 2nd stimuli produce no twitch—90% blockade
  - If 1st stimuli produce no twitch—100% blockade

#### **Return of all four twitches is complete reversal of neuromuscular blockade.**

- **Site:** Ulnar N/orbicularis oris muscle
- **CNS monitoring** [Bispectral index (BIS)]
  - Advancement in EEG monitoring to monitor awareness during surgery amongst other uses
  - It uses bifrontal recordings of EEG and uses burst suppression ratio, relative alpha: beta ratio and bicoherence values to calculate BIS of the patient based on the data extrapolated from 100s of database EEGs

- Results range from 0 to 100 whereby 0 = complete suppression of activity (isoelectric EEG) and 100 = full awareness
- Help in determining level of patient's awareness during surgery, lowers the consumption of anesthetics during surgery, helps in earlier awakening and faster recovery from anesthesia
- Also helps in titration of levels of sedative medications
- BIS < 40 for > 5 minutes is an independent predictor of increased perioperative morbidity due to stroke and myocardial infarction in high-risk patients.

**Q57. Enumerate the causes of Postoperative fever. Discuss its management.**

**Ans.** Temperature >38.3°C in postoperative period

**Causes**

6 W s	Cause	Post operative Day
<b>Wind</b>	Atelectasis	1
	Pneumonia	2
<b>Water</b>	Urinary tract infection	2
	Thrombophlebitis	2
<b>Wound</b>	Wound infection	3,4
	Anastomotic leak	5-8
<b>Walk</b>	Deep vein thrombosis	5-8
	Pulmonary embolism	5-8
<b>Wonder drugs</b>	Anesthetic drugs	Immediate/intraoperative
	Antibiotics	Anytime
	Blood products	Anytime
<b>Waste products</b>	GIT	Anyday after 3rd day

**Immediate**

Fever that occurs in operation theater or in immediate postoperative period

*Causes*

- Malignant hyperthermia
- Medication (antibiotics, blood transfusion)
- Necrotizing infections (*Clostridium*, group A *Streptococcus*)

**Acute fever**

Fever in the first week after operation

*Causes*

- Atelectasis, pneumonia
- Urinary tract infection (catheter >2 days increases the risk)
- IV line infection
- Wound infection, anastomotic leak
- Pulmonary embolism, deep vein thrombosis
- Myocardial infarction, pancreatitis, aspiration pneumonia.

**Subacute fever ( > 1 week)**

- UTI, IV line
- SSI
- Antibiotic induced fever
- Pseudomembranous enterocolitis
- Febrile drug reactions.

**Management**

- Appropriate resuscitative measures and care of patient's airway, breathing and circulation
- Cultures from all existing lines, sputum, urine, blood and wound in case of SSI
- Remove/replace peripheral lines, nontunneled lines and catheter
- Chest X-ray, ultrasound for wound and intra-abdominal collections
- Evaluation of drug list
- Lower limb doppler and chest CT, D-dimer for suspected PE/DVT
- Open/debride/drain wound in case of SSI
- Start broad spectrum antibiotics in case of pneumonia, bacteremia, UTI, sepsis and continue for 10 to 14 days except in septic thrombosis or endocarditis where antibiotics need to be continued for 4–6 weeks.

**Catheter salvage therapy**

- Indicated in patients with tunnelled catheters that are too risky to remove or replace or in patients with coagulase negative staphylococci with no evidence of metastatic disease/severe sepsis/persistent bacteremia/tunnel infection
- Achieved by antibiotic lock therapy in which, the catheter is filled with antibiotic solution for few hours every day.

**Q58. Discuss the role of prophylactic antibiotics in surgery.**

**Ans.**

**Definitions**

- **Infection:** The invasion of the body by pathogenic microorganisms that reproduce and multiply causing disease by local cellular injury, secretion of a toxin or antigen-antibody reaction in the host
- **Colonization:** The presence of bacteria on a body surface (like on the skin, mouth, intestines or airway) without causing disease in the person
- **Prophylactic antibiotic treatment:** The use of antibiotics before, during or after a diagnostic, therapeutic or surgical procedure to prevent infectious complications
- **Therapeutic antibiotic treatment:** The use of substances that reduce the growth or reproduction of bacteria, including **eradication therapy** (Antimicrobial therapy prescribed to clear infection by an organism or to clear an organism that is colonising a patient but is not causing infection)
- **Primary prophylaxis** refers to the prevention of an initial infection
- **Secondary prophylaxis** refers to the prevention of recurrence or reactivation of a pre-existing infection
- **Eradication** refers to the elimination of a colonized organism to prevent the development of an infection.

**Need of prophylaxis**

- Prevent surgical site infection (SSI)
- Prevent SSI-related morbidity and mortality
- Reduce the duration of hospital stay and cost of health care

At the same time care should be taken so that there is

- Minimal effect of antibiotics on the patient's normal bacterial flora
- Minimal adverse effects of the used drugs
- Minimal change to the patient's host defences

**Risks**

- Unknown allergy to drugs and anaphylaxis (mainly penicillin and cephalosporin group)
- Antibiotic associated diarrhea (even single dose can cause it)
- Antibiotic resistance
- Multiresistance carriage (mainly MRSA and VRE).

**Choice of Antibiotics**

- **Must cover the expected pathogens**
- Local resistance patterns
- **Narrow spectrum, less expensive**
- Carriers of **MRSA** (anterior nasal swab cultures) should have a course of eradication therapy prior to high risk surgery (*orthopaedic implant, heart valve, vascular graft or shunt or CABG*).

**Dosage and timing of administration**

- Serum and tissue concentrations exceeding the minimum inhibitory concentration (MIC)
  - At the time of incision
  - For the duration of the procedure.
- Intravenous route
- **Should be given ≤60 minutes** before the skin is incised
- For fluoroquinolones and vancomycin, the administration should begin within 120 minutes before surgical incision
- A **single standard therapeutic dose** of antibiotic is enough
- Arthroplasty—24 hours
- Cardiothoracic surgery—48 hours.

**Need of re-administration**

- The **redosing interval** should be measured from the time of administration of the preoperative dose
- Duration of surgery longer than twice the half-lives of the drug
- **Major intraoperative blood loss**
  - In adults (>1,500 mL)
  - In children (25 mL/kg)

Additional dosage of prophylactic antibiotics should be considered after fluid replacement.

**Antimicrobial prophylaxis for the prevention of infective endocarditis**

- **Evidence is weak and inconclusive**
- The 2007 AHA guideline. Similar indications by the British Society for Antimicrobial Chemotherapy
- **The highest risk conditions** (Prosthetic heart valves, a prior history of IE, unrepaired cyanotic congenital heart disease).
- **The following are the highest risk procedures** (All dental procedures, procedures in patients with ongoing GI or GU tract infection, on infected skin, skin structure, or musculoskeletal tissue and cardiac surgery).
- **The antibiotics should be procedure and patient specific**
- **Antibiotic of choice—oral amoxicillin**
- **Penicillin allergy—ceftriaxone, cefazolin, vancomycin, clindamycin**

**Important considerations**

- Agents that are FDA-approved for use in surgical antimicrobial prophylaxis include cefazolin, cefuroxime, ceftiofex, cefotetan, ertapenem, intranasal mupirocin and vancomycin
- The safety and efficacy of topical antimicrobials have not been clearly established.

**Recommendations in surgery**

Antibiotic prophylaxis is recommended in clean surgery with prosthesis, all clean and contaminated surgeries and in all dirty surgeries along with therapeutic antibiotic therapy.

**Q59. Discuss probiotics, prebiotics and synbiotics.**

Ans.

**Definitions**

- **Probiotics:** Live microorganisms which, when administered in adequate amounts, confer a health benefit on the host
- **Prebiotics:** Nondigestible substances that provide a beneficial physiological effect for the host by selectively stimulating the favorable growth or activity of a limited number of indigenous bacteria
- **Synbiotics:** Products that contain both probiotics and prebiotics
- Species of *Lactobacillus* and *Bifidobacterium* are most commonly used as probiotics but the yeast *S. cerevisiae* and some *E. coli* and *Bacillus* species are also used as probiotics. Lactic acid bacteria, including *Lactobacillus* species, which have been used for preservation of food by fermentation for thousands of years can serve a dual function by acting as agents for food fermentation and in addition, potentially imparting health benefits.

**Benefits of using probiotics/prebiotics***Probiotics*

- **Immunologic benefits**
  - Activate local macrophages to increase antigen presentation to B lymphocytes and increase secretory immunoglobulin A (IgA) production both locally and systemically
  - Modulate cytokine profiles
  - Induce hyporesponsiveness to food antigens.

- **Nonimmunologic benefits**

- Digest food and compete for nutrients with pathogens
- Alter local pH to create an unfavorable local environment for pathogens
- Produce bacteriocins to inhibit pathogens
- Scavenge superoxide radicals
- Stimulate epithelial mucin production
- Enhance intestinal barrier function
- Compete for adhesion with pathogens
- Modify pathogen-derived toxins.

*Prebiotics*

- Metabolic effects: Production of short-chain fatty acids, fat metabolism, absorption of ions (Ca, Fe, Mg)
- Enhancing host immunity (IgA production, cytokine modulation, etc.).

**Clinical applications**

*Colon cancer*

- Studies suggest that a synbiotic preparation can decrease the expression of biomarkers for colorectal cancer.

*Diarrhea*

- **Treatment of acute diarrhea:** Different probiotic strains such as *L. reuteri*, *L. rhamnosus* GG, *L. casei*, and *S. cerevisiae* (boulardii) are useful in reducing the severity and duration of acute infectious diarrhea in children. The oral administration of probiotics shortens the duration of acute diarrheal illness in children by approximately 1 day.
- **Antibiotic-associated diarrhea:** In antibiotic-associated diarrhea, there is strong evidence of efficacy for *S. boulardii* or *L. rhamnosus* GG in adults or children who are receiving antibiotic therapy.
- **Radiation-induced diarrhea:** *L. casei*, *L. plantarum*, *L. acidophilus*, *L. delbrueckii*, *B. longum*, *B. breve*, *B. infantis*, and *S. thermophilus* are effective in the treatment of radiation-induced diarrhea.

*Hepatic encephalopathy*

- Prebiotics such as lactulose are commonly used for the prevention and treatment of this complication of cirrhosis. Minimal hepatic encephalopathy was reversed in 50% of patients treated with a synbiotic preparation (four probiotic strains and four fermentable fibers including inulin and resistant starch) for 30 days.

*Pouchitis*

- There is good evidence for the usefulness of probiotics in preventing an initial attack of pouchitis and in preventing further relapse of pouchitis after the induction of remission with antibiotics. Probiotics can be recommended to patients with pouchitis of mild activity or as maintenance therapy for those in remission.

*Ulcerative colitis*

- The probiotic *E. coli* Nissle strain may be an equivalent to mesalazine in maintaining remission of ulcerative colitis. There is an inadequate research evidence to be certain that other probiotic preparations are effective in ulcerative colitis.

*Crohn's disease*

- Studies of probiotics in Crohn's disease have been disappointing and a recent Cochrane systematic review concluded that there is no evidence to suggest that probiotics are beneficial for maintenance of remission in Crohn's disease.

*Irritable bowel syndrome (IBS)*

- Several studies have demonstrated significant therapeutic gains with probiotics in comparison with placebo. A reduction in abdominal bloating and flatulence as a result of probiotic treatments is a consistent finding in published studies. Some strains may ameliorate pain and provide global relief in addition. *Lactobacillus reuteri* may improve colicky symptoms within one week of treatment as shown in a recent trial with 90 breast-fed babies with infantile colic. In summary, there is literature suggesting that certain probiotics may improve the principal symptoms in persons with IBS.

*Lactose malabsorption*

- *S. thermophilus* and *L. delbrueckii* subsp *bulgaricus* improve lactose digestion and reduce symptoms related to lactose intolerance.

*Necrotizing enterocolitis*

- Clinical trials have shown that probiotic supplementation reduces the risk of necrotizing enterocolitis in preterm neonates of less than 33 weeks' gestation. A systematic review of randomized controlled trials also indicated a reduced risk of death in probiotic treated groups. In summary, there is strong support for the use of certain probiotic strains in preterm infants.

## **Q60. What is surgical site infection? Discuss the methods to reduce surgical site infection.**

### **Enumerate the factors responsible for surgical site infection.**

#### **Ans. Definition**

*Nosocomial Infection*

- An infection acquired in hospital by a patient who was admitted for a reason other than that infection
- An infection occurring in a patient in a hospital or other health care facility in whom the infection was not present or incubating at the time of admission. This includes infections acquired in the hospital but appearing after discharge and also occupational infections among staff of the facility.

Infections occurring for more than 48 hours after admission are usually considered nosocomial. Amongst surgical patients, SSI are the most common nosocomial infections (38%).

**Classes of SSI***CDC criteria for defining a surgical site infection*

- **Superficial incisional SSI:** Infection occurs within 30 days after the operation and infection involves only skin of subcutaneous tissue of the incision *and at least one of the following:*
  - Purulent drainage with or without laboratory confirmation from the superficial incision
  - Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision

- At least one of the following signs or symptoms of infection:
  - Pain or tenderness
  - Localised swelling
  - Redness
  - Heat
  - And superficial incision deliberately opened by a surgeon, unless incision is culture negative
- Diagnosis of superficial incisional SSI by the surgeon or attending physician.
- **Deep incisional SSI:** Infection occurs within 30 days after the operation if no implant is left in place or within one year if implant is in place and the infection appears to be related to the operation and infection involves deep soft tissues (e.g. fascial and muscle layers) of the incision and at least one of the following:
  - Purulent drainage from the deep incision but not from the organ/space component of the surgical site
  - A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms:
    - Fever ( $>38^{\circ}\text{C}$ )
    - Localized pain
    - Tenderness unless site is culture-negative
  - An abscess or other evidence of infection involving the deep incision is found on direct examination, during re-operation or by histopathological or radiological examination
  - Diagnosis of deep incisional SSI by a surgeon or attending physician.
- **Organ/space SSI:** Infection occurs within 30 days after the operation if no implant is left in place or within one year if implant is in place and the infection appears to be related to the operation and infection involves any part of the anatomy (e.g. organs or spaces), other than the incision, which was opened or manipulated during an operation and at least one of the following:
  - Purulent discharge from a drain that is placed through a stab wound into the organ/space
  - Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space
  - An abscess or other evidence of infection involving the organ/space that is found on direct examination, during re-operation, or by histopathologic or radiological examination
  - Diagnosis of an organ/space SSI by a surgeon or attending physician.

### Strategies to prevent SSI

#### *Objectives*

- Reduce the inoculum of bacteria at the surgical site
- Surgical site preparation
- Antibiotic prophylaxis strategies
- Optimize the microenvironment of the surgical site
- Enhance the physiology of the host (host defenses)



In relation to risk factors, classified as:

- Patient-related (intrinsic)
- Preoperative
- Operative

#### **Patient-related factors**

- **Diabetes**—recommendation
- Preoperative
- Control serum blood glucose—reduce HbA1C levels to <7% before surgery if possible
- Postoperative (cardiac surgery patients only)
- Maintain the postoperative blood glucose level at less than 200 mg/dL
- Other patient related factors include:
  - Smoking, anemia, malnutrition
  - Hypoalbuminemia, jaundice
  - Obesity, hyperlipidemia
  - Ascites, PVD
  - Immunosuppression.

#### **Procedure-related risk factors**

- **Hair removal technique (clipping > on table shaving > previous night shaving)**
- Preoperative infections control and bath
- Surgical scrub
- Skin preparation
- Antimicrobial prophylaxis
- Surgeon skill/technique/instruments
- Asepsis
- Operative time (should be within 1.5 times the normal)
- Operating room characteristics/OT sterility.

#### **Surgeon skill and technique**

Excellent surgical technique reduces the risk of SSI

*Includes*

- Gentle traction and handling of tissues
- Effective hemostasis
- Removal of devitalized tissues
- Obliteration of dead spaces
- Irrigation of tissues with saline during long procedures
- Use of fine, nonabsorbed monofilament suture material
- Wound closure without tension.

#### **Examples of multimodal approach(es) to reduce SSI**

- Timely antibiotic prophylaxis, strict glycemia control, no shaving  
SSI 1.5% vs. 3.5% in controls
- 100k lives campaign  
(antibiotic prophylaxis, glycemia control, normothermia)

SSI from 2.3% to 1.7% (-27%)

- Risk stratification of patients according to National nosocomial infection surveillance system (NNISS) include wound type (contaminated/dirty), ASA grade (3,4,5) and duration of operation (>75th percentile of normal) to give risk of SSI in a particular patient.
- Bowel preparation lowers the patient's risk of infection from that of a contaminated case (25%) to a clean contaminated case (5%).

## SURGERY OF THE SALIVARY GLANDS

**Q61. Write the classification of salivary gland neoplasms.**

**Ans.**

**Classification of salivary gland neoplasms is as follows:**

<b>Epithelial</b>	<b>Benign</b> <ul style="list-style-type: none"> <li>• Pleomorphic adenoma</li> <li>• Monomorphic adenoma—oxyphil adenoma, Warthin tumor or adenolymphoma</li> <li>• Papilloma—intraductal or inverted.</li> <li>• Basal or canalicular adenoma</li> </ul> <b>Malignant</b> <ul style="list-style-type: none"> <li>• Mucoepidermoid carcinoma</li> <li>• Adenoid cystic carcinoma</li> <li>• Malignant pleomorphic adenoma</li> <li>• Acinic cell tumor</li> <li>• Papillary or mucinous adenocarcinoma</li> <li>• Oncocytic carcinoma</li> <li>• Malignant mixed tumor</li> </ul>
<b>Mesenchymal</b>	<ul style="list-style-type: none"> <li>• Hemangioma</li> <li>• Neurofibroma</li> <li>• Lymphoma</li> <li>• Lymphangioma</li> </ul>
<b>Metastatic</b>	<ul style="list-style-type: none"> <li>• Malignant melanoma</li> <li>• Epidermoid carcinoma</li> </ul>

**Q62. Write a note on pleomorphic adenoma.**

**Ans.**

### **Introduction**

- It is the most common neoplasm of the salivary gland
- It is most commonly found in the superficial lobe of parotid gland
- Commonly affects women in middle age group
- Can be benign or malignant.

### **Pathology**

- Contains both epithelial and mesenchymal components and therefore is called pleomorphic

- Unicentric origin
- Recurrences are multicentric
- Usually encapsulated
- Has finger like projections into surrounding.

**Clinical features of benign adenoma**

- Females of middle age group present with a painless slowly enlarging swelling behind the ear lobule in the parotid region and raising the ear lobule present since many years
- It is a mobile, nontender, rounded swelling with well defined margins and smooth surface and firm to hard consistency
- Facial nerve is not involved and overlying skin is free
- No lymphadenopathy
- A pleomorphic adenoma of deep lobe may push tonsil medially and be palpable intraorally.

**Clinical features suggestive of malignancy**

- Rapid increase in size
- Pain and tenderness
- Lymphadenopathy
- Fixity to skin or deeper structures
- Facial nerve involvement
- Multicentric tumors suggest recurrence.

**Investigations**

- FNAC is enough for diagnoses
- CT head and neck or MRI in case of malignancy
- Preoperative workup for anesthetic fitness.

**Treatment**

- Superficial parotidectomy (**Patey's operation**)
- Total parotidectomy without facial nerve preservation for malignancy.

**Q63. Write a note on Warthin tumor.****Write a note on adenolymphoma.****Ans. Introduction**

- It is the second most common neoplasm of the salivary gland
- It is found only in the superficial lobe at lower portion of parotid gland
- Commonly affects males in old age group
- It is always benign.

**Pathology**

- Smoking is a risk factor
- Contains both epithelial and lymphoid components and therefore is called adenolymphoma
- Unicentric origin
- Usually well encapsulated

- Also contains cystic areas and papillary epithelial projections with double layer of lining epithelium and therefore is also called papillary cystadenoma lymphomatosum.

**Clinical features**

- Males of old age group present with a painless slowly enlarging swelling behind the ear lobule in the parotid region and raising the ear lobule present since many years
- It is a mobile, nontender, rounded swelling with well defined margins and smooth surface and soft to cystic consistency
- Facial nerve is not involved and overlying skin is free
- No lymphadenopathy.

**Investigations**

- FNAC is enough for diagnoses
- Shows hot spot on technetium pertechnetate scan
- Preoperative workup for anesthetic fitness.

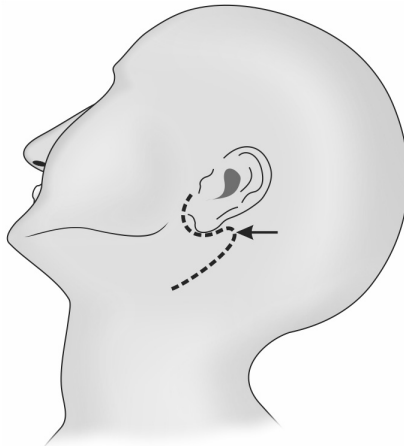
**Treatment**

- Superficial parotidectomy (**Patey's operation**).

**Q64. Write a note on superficial parotidectomy and enumerate its complications.**

**Ans.** Superficial parotidectomy is the removal of the superficial lobe of parotid gland and is indicated in all the benign tumors of the superficial lobe of the parotid gland.

- Incision
  - Blair incision
  - Modified Blair incision
  - Sistrunk incision



**Fig. 2:** Incision for parotid surgery

- The marking starts from the preauricular region, goes upto mastoid tip from below the ear, then comes anteriorly to merge with normally present neck line below mandible
- When the angle at mastoid tip between horizontal and vertical limbs of the incision is pointed, it is called as Blair incision. This pointed tip is associated more with chances of necrosis and therefore modified blair incision is used now which has made that angle curved

- After the incision, the anterior flap is raised superficial or deep to platysma to expose the anterior parotid surface covered by deep fascia, posterior flap raised upto sternomastoid and superiorly upto the junction of cartilaginous and bony auditory tube
- During raising the anterior flap, care should be taken to avoid injury to facial nerve branches as they emerge from the parotid gland anteriorly
- Next, the deep fascia is incised along the mastoid tip and posterior belly of digastrics identified to begin the dissection at the posteroinferior border of the parotid gland and proceed anterosuperiorly
- The dissection is done in the ptery's fasciovenous plane with the nerve and vein lying in it and the arteries deeper to this plane
- The tragal pointer cartilage is 1 cm above and superficial to the posterior belly of digastric and aids in identification of the facial nerve trunk. The facial nerve lies 1 cm medial and inferior to the tragal pointer
- The retromandibular vein, styloid process and tympanomastoid sutures are also useful landmarks to identify the facial nerve in this region. The nerve is superficial to retromandibular vein, lateral to styloid process and just inferior to the tympanomastoid suture line
- The nerve enters the parotid at its posteromedial surface
- The technique of parotid dissection involves inserting the blade of instrument along the direction of nerve, lift, spread and then cut in that order
- The superficial lobe of the parotid gland is resected keeping the deep lobe, parotid duct and all the facial nerve branches in situ
- The incision is closed with sutures and drain placed which is kept for around 2 days. The sutures are removed on 5th day usually to decrease scarring.

### Complications

#### *Wound complications*

- Seroma
- Hematoma
- Infection and stitch line abscess
- Flap necrosis

#### *Facial nerve paresis or paralysis*

#### *Parotid fistula*

*Sensation loss at an angle of mandible due to greater auricular nerve damage.*

#### *Frey syndrome*

- Causes damage to auriculotemporal nerve during surgery followed by cross connection between parasympathetic fibres of auriculotemporal nerve and sympathetic fibers of the sweat glands supplying the angle of jaw
- This leads to sweating at parotid region and angle of jaw when the patient eats instead of salivary secretion
- Minor's starch iodine test is confirmatory
- Management
  - **Prevention** by taking a segment of auriculotemporal nerve during surgery to prevent regeneration

**– Treatment**

- Local aluminium chloride application
- Tympanic neurectomy (surgical division of the cross connected fibers)
- Botulinum toxin injection.

## MISCELLANEOUS GENERAL SURGERY TOPICS OF IMPORTANCE

**Q65. Write a note on surgical audit.**

**Ans. Audit means**

- Outcomes measurement
- Quality of services analyses
- Improvement in services based on the audit reports

**Aims**

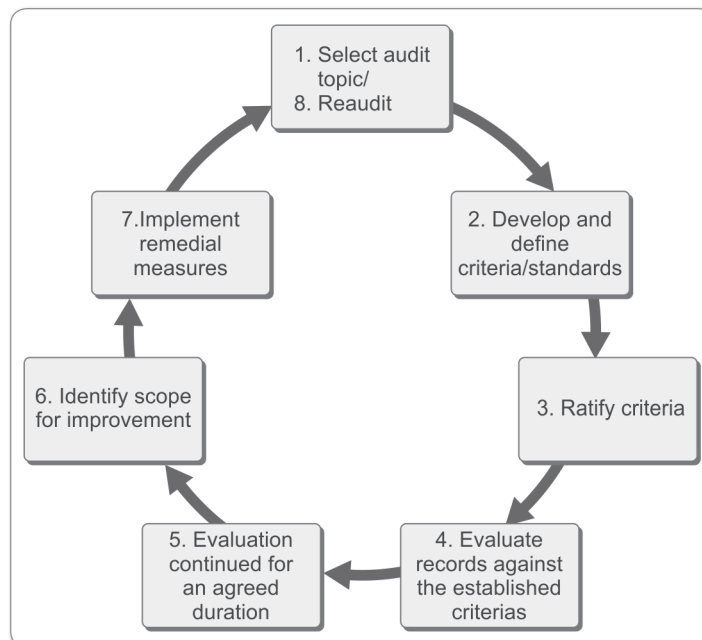
- Allows the surgeon to assess his skills and level of care against the present standards
- Helps in giving the best and required care to the patient

**Types**

- Hospital/institutional audit
- National audit.

Issues can relate to many diverse problems or quality of care that surgeon delivers to the patient.

Audit is a continuous process and can be summarized by the following audit cycle:



**Audit topic can be**

- A physical sign or symptom
- A screening procedure
- A diagnostic test
- A diagnoses
- A treatment procedure
- A follow-up procedure

**Criteria** that are defined should be (Mnemonic: CARES UFO)

- Comprehensive
- Acceptable
- Relevant
- Explicit
- Specific
- Uniform
- Feasible
- Objective and verifiable.

Ratification of criteria comes next which means passing the criteria to all the participating members of audit and seeing that the criteria are approved by all those involved.

**Evaluation of records**

- OT registers
- Health workers diary
- Follow-up register
- Medical records department

Once all this is done, evaluation and identification of remedial measures is done and remedial measures are implemented.

After implementation of remedial measures, check for compliance to new measures and if there is non compliance, do re-audit of noncompliant measures within 3 months of the implementation.

The same cycle continues as audit is a continuous process.

**Q66. Discuss ethics and its impact on patient management.**

**Ans.**

**Definition**

**Medical ethics** is a system of moral principles that apply values and judgments to the practice of medicine.

**Four pillars of medical ethics**

- **Autonomy:** This is the right of the patient to make informed decisions on his own will
- **Beneficence:** The doctor should always act for the betterment or welfare of the patient
- **Nonmaleficence:** The doctor should not indulge in any action that can intentionally harm the patient
- **Justice:** Justice is a complex ethical principle with meanings that range from the fair treatment of individuals to the equitable allocation of healthcare finance and resources.

Justice is concerned with the equitable distribution of benefits and burdens to individuals in hospitals or social institutions and how the rights of various individuals are realized.

It is important to remember that **CONFIDENTIALITY is NOT a pillar of medical ethics.**

This is an important issue because confidentiality is confirming to all the four pillars of medical ethics and is therefore considered important ethical principle to follow.

However, it is not an ethical pillar because in some situations, confidentiality may need to be broken as in following:

- To protect harm to third party
- To prevent crime
- In court of law for justice
- Fitness to drive
- Fitness to work where others might be affected

Tools of practice for medical ethics include 4 important components and 4 levels of each component with are mentioned below:

- **Competence**
  - Expert competence
  - Shared competence
  - Team competence
  - Not competent
- **Resources**
  - Equal distribution of adequate resources
  - Adequate distribution possible with rationing
  - Scarce resources
  - Inadequate resources
- **Abiding by law**
  - Action is completely protected by law
  - Reasonably predictable law
  - Civil law not protecting the action
  - Illegal act
- **Morality**
  - Uncontrovertially moral
  - Acceptable moral
  - Morally doubtful
  - Immoral

This means that an act by a fully competent person with adequate utilization of resources which is legally and morally appropriate for a given patient is an ethical act.

The consequence evaluation of such acts is done using the individual, family and society tool to look at the consequences of an ethical or unethical act on both the patient and the doctor side.

### Implementation

- Only ethical acts are the acts of duty for a doctor
- It is a doctor's duty to:



- Follow confidentiality issues
- Provide morally and legally correct and competent healthcare to patient
- Protect the rights of the third party

This emphasis on duty is part of an area of ethics called **Deontology**.

**Q67. Write a note on universal precautions.**

**Enumerate the standard precautions used in surgeries to prevent against HIV.**

**Ans.**

- Center for disease control in Atlanta gave the universal precautions in 1987 to minimize the risk of transmission of blood-borne infections
- In 1996, CDC renamed it as standard precautions to include universal precautions and body substances isolation to prevent risk of infection transmission from almost all body fluids and tissues
- These apply to all body fluids such as blood, body secretions, all body fluids such as semen, vaginal secretions, CSF, urine, etc. organs or tissues, cell cultures or tissue cultures, organ cultures, animal parts or fluids, non-intact skin and mucous membranes.

**These precautions include:**

- **Proper handwashing**
  - Before and after coming in contact with any patient
  - Before and after coming in contact with body fluids or specimens
  - Before and after glove wearing anytime
  - Steps are given to standardize the technique and prevent infection transmission
- **Wearing double pair of gloves:** Single pair does not limit risk as much as double pair while three pairs give no added benefit
- **Caps and masks**
- **Apron**
- **Protective equipment for eyes and feet:** Eye protection equipment include plastic glasses with side shields, goggles, chin length masks and masks with visors. The gum boots or canvas shoes are for feet protection especially while doing TURP like procedures
- **Prevention of needle stick injury should be done by following measures:**
  - No more than single pair of hands should work in an operation field.
  - Use instruments to handle needles.
  - Always put sharps in a tray rather than then directly.
  - Direct sharp instruments should be kept away from the assistant's hands.
  - Never directly recap a needle with two hands.
  - Discard sharps with caution. Never bend the needle with hands.
  - Always use puncture proof containers.

This is important because the risk of transmission due to needle sticks is 30% for HBV, 3% for HCV and 0.3% for HIV.

**Q68. What is a biopsy? What are its types?**

**Discuss the role of biopsy in surgery.**

**Ans.** Biopsy is a procedure to obtain tissue for microscopical examination, usually to perform a diagnosis.

**Indications for biopsy**

- Any lesion or swelling in skin, subcutaneous tissue, muscle or organs such as liver, lung, tongue, rectum, bone marrow, prostate, brain, kidney and almost all organs in the body before excision are meant for definitive diagnoses.
- Any lesion suspicious of malignancy (Persistence of lesion or ulcer, sudden increase in size, change in character, color or consistency, bleeding to touch, becomes fixed to surrounding structures)
- Precancerous lesions (Leukoplakia, etc.)
- Inflammatory lesions: Ulcerative colitis, Crohn's disease, amyloidoses, vasculitis, etc.
- Bone lesions not identified by clinical and radiographic findings
- Nonhealing ulcers.

**Types of biopsy**

- Aspiration biopsy cytology or fine needle aspiration cytology (**Discussed in previous question**).
- Trucut biopsy
- Incisional biopsy—wedge biopsy or punch biopsy
- Excisional biopsy.

**Trucut (core needle) biopsy**

- It is the aspiration biopsy using trucut needle with 20 mm specimen notch and centimeter depth markings that assist in depth perception and sharp cutting edge which help in cutting, high quality specimen cleanly.
- It can be done directly by palpation or under ultrasound, MRI or stereotactic guidance or vacuum assisted core biopsy.
- **Indications**
  - Breast lesions, liver lesions, bone lesions are common indications
- The histological architecture of the tissue is retained here as it is not aspiration but rather a type of core needle incision biopsy wherein cylinders or cores of tissues are removed and not cells alone.

**Incisional biopsy**

- The intent of an incisional biopsy is to sample a portion of the lesion
- If the lesion is large, more than one area may require sampling
- **Indications**
  - In all the lesions which are very large to directly undergo excisional biopsy or where excision biopsy can produce functional limitations not justified for all diagnoses of the lesion, incisional biopsy is to be done first to confirm the diagnoses.
  - For example, extremity soft tissue sarcomas.
- **Technique**
  - A wedge resection of the lesion is done such that margin extend into the normal tissue on deep surface and sometimes also on the lateral surface
  - Necrotic tissue should be avoided.

**Punch biopsy**

- Can be used for incisional or excisional biopsy .

- **Technique**
  - Biopsy punches should range in size from 2 to 10 mm in diameter and the residual wound should be allowed to heal by secondary intention.
- **Disadvantages**
  - It is difficult to obtain adequate, representative tissue deeper than the superficial lamina propria.

**Brush biopsy**

- Firm pressure with a circular brush is applied, rotated for about ten times to cause an abrasion
- The cellular material picked up by the brush is transferred to a glass slide, preserved and dried.

**Excisional biopsy**

- **Indications:**
  - Used for small lesions
  - When complete excision with a margin of normal tissue is possible without functional debility or amputation.
- **Technique**
  - Complete removal of the lesion with 2–3 mm normal tissue surrounding the lesion is performed
  - Direct infiltration of anesthesia into the lesion and use of electrocautery near the lesion should be avoided
  - The specimen should be immediately placed and completely immersed in 10% formalin solution after orientation to allow determination of the right, left, superior, deep and inferior surfaces of the specimen
  - Primary closure of the wound is usually done.

**Q69. Write a note on fine needle aspiration cytology and its role in surgery.****What is aspiration biopsy cytology? Discuss its uses.**

**Ans.** FNAC is an interventional cytology technique.

**Indications**

- Diagnoses of palpable mass lesions such as in breast, lymph nodes, thyroid, soft tissue lesions etc.
- Diagnoses of nonpalpable mass lesions is also done using FNAC under assistance of radiological techniques such as ultrasound, CT scan, MRI, stereotactic localization and FNAC or endoscopy guided FNAC
- Salivary gland FNAC, diagnoses of abdominal lesions, liver or lung lesions, prostate, retroperitoneal lumps, etc. are also diagnosed using FNAC.

**Technique**

- A syringe with 22 gauge needle is used for performing FNAC. Glass slides and suitable fixative are also required for FNAC
- The procedure is usually done with or without local anesthesia
- 6–10 needle passes are made to collect adequate sample
- The cells collected are smeared on glass slides and prepared smears are wet fixed or air dried or both

- Wet fixation using Hematoxylin and Eosin stain is mainly used to study nuclei and nuclear features whereas air dried smears stained with Giemsa or Leishman stain are used to study cytoplasmic components and background stromal details.

### Advantages

- It is an outpatient procedure and no hospitalization is required
- It is painless and rapidly carried out
- It can be done without anesthesia
- It is less traumatic than biopsy
- It is cheap and safe
- Result is obtained rapidly.

### Complications

- Infection
- Bleeding
- Pneumothorax for lung or pleural FNAC
- Bile leakage or bleeding in liver FNAC
- Pancreatitis after pancreas FNAC.

### Disadvantages

- It is cytology, therefore, cannot differentiate with certainty between in situ and malignant lesions
- Requires experienced cytopathologists to make the reports
- In thyroid, cannot differentiate between follicular adenoma and carcinoma
- Immunohistochemical markers are not routinely available to be carried out on FNAC specimens
- Inadequate sample can result in false negative diagnoses. Therefore, a negative FNAC alone is not enough to rest assured if malignancy was suspected before the procedure and it needs to be repeated or biopsy done to establish the diagnoses with certainty.

## Q70. Write a note on sterilization techniques.

**What is sterilization? What is disinfection? Enumerate the techniques of sterilization.**

**Ans. Sterilization:** Killing of all the microorganisms, either in the vegetative or spore state

**Disinfection:** Killing of all the pathogenic organisms but not the spores

**Antisepsis:** Prevention of infection on skin and mucous membrane.

### Techniques of sterilization and disinfection

Sunlight	UV rays	It is natural sterilization method
Dry heat—act by free radical mediated damage and protein denaturation	Red heat	
	Flaming	Tip of forceps
	Incineration	All pathological wastes Dirty dressings
	Hot air oven	160°C at 15 lb pressure for 1 hr Used for syringes, oils and paraffin, glassware and powders

Contd...

Contd...

Sunlight	UV rays	It is natural sterilization method
<b>Moist heat</b>	Pasteurization	Milk
	Inspissation	80–85°C for 1 hr on 3 consecutive days Used for Lowenstein-Jensen medium and Loeffler's media.
	Steam at 100°C (Tyndallization)	100°C for 20 minutes on 3 consecutive days For media containing serum, egg or gelatine.
	Autoclave	Steam at 121°C for 15–20 minutes Used for surgical equipment, pharmaceuticals, dressings. Bacillus stearothermophilus or bacillus subtilis is used as control
<b>Radiation</b>	Ionising (X-rays, Gamma rays)	Plastic material (disposable syringes, swabs, catheters, chest tubes, culture plates, ryles tube)
	Non ionising (Infrared and UV rays)	Used for operation theatre sterilization
<b>Filtration</b>	Kieselghur type or Kaolin and sand type/ sintered glass filter/ membrane filter	Used for antibiotic solution, serum or solutions containing sugar and gelatin
<b>Chemical</b>	Alcohol	Ethyl alcohol is used as skin antiseptic
	Aldehyde	Fumigation
	2% glutaraldehyde (Cidex) for 20 minutes for disinfection	Rubber equipment, laparoscopy instruments, cystoscopy instruments, all endoscopes, endotracheal tube and oxygen mask
	Chlorhexidine (Phenol group)	Skin antiseptic
	Ethylene oxide	All tubings and presterilized catheters are sterilized by ethylene oxide or gamma rays All heart lung machines, respirators, suture materials, disposable syringes are presterilized by ethylene oxide
	Formaldehyde gas	Fumigation of operation theatre
	Betapropiolactone	Better agent than formaldehyde
	Cationic or anionic surface active agents (Cetavlon)	Used in antiseptic soaps

**Q71. Write a note on hospital waste management.****Ans.**

- **Biomedical waste is defined as** any waste which is generated during diagnoses, treatment or immunization of human beings or animals or in research or production or testing of biologicals

- Of the biomedical waste generated nearly 70–80% is nonhazardous but the remaining 10–15% is hazardous and can cause health issues if it is not properly disposed off
- The hazards are both to generators and to the patients and can cause infections, injury from sharps, radiation hazards, burns, fires and many more
- **Options for destruction of the waste is through one of the following techniques:**
  - Incineration mainly for solid wastes and soiled wastes
  - Wet and dry thermal treatment
  - Microwave irradiation
  - Landfill
  - Chemical disinfection using 1% hypochlorite solution mainly for liquid wastes such as blood, urine, stools or hospital sewage
- Different containers are provided for the waste segregation according to its disposal. These containers and their colour coding are government mandated and routine checks are done to ensure the enforcement of the rules of waste disposal at hospital level
- It is said that the waste segregation should begin at the site of generation and thus this colour coding is very essential to remember. These bags are then collected by the waste disposal teams and disposed appropriately.

**The color coding is as follows:**

<b>Red bag or disinfected container:</b> Autoclaving or chemical disinfection	<ul style="list-style-type: none"> <li>• Contaminated items with blood and/or body fluids.</li> <li>• Waste from laboratories, microorganisms specimens, research wastes such as cell cultures, wastes produced during pharmaceutical production of drugs, toxins, biological or cultures</li> <li>• Tubings, catheters, IV sets, blood transfusion bag and sets, Urobag</li> </ul>
<b>Blue puncture proof container or Blue plastic bag:</b> Chemical treatment and destruction or autoclaving and destruction or shredding	<ul style="list-style-type: none"> <li>• Needles, syringes, scalpel blades, glass bottles, broken ampoules that can cause injury</li> </ul>
<b>Yellow plastic bag:</b> Incineration or landfill burial	<ul style="list-style-type: none"> <li>• Human tissues, organs, animal tissues and organs, body parts, human or animal waste from hospitals, colleges or animal houses</li> <li>• Waste food stuff from hospital, fecal, urine soiled stuff</li> </ul>
<b>Black plastic bag:</b> Landfill Burial	<ul style="list-style-type: none"> <li>• Empty plastic bottles, coverings of disposable tubings, noncontaminated plastic waste</li> <li>• Outdated, discarded medicines</li> <li>• Chemicals used in production of biological, disinfectants or insecticides</li> </ul>

### **Q72. Enumerate the complications of tracheostomy.**

**Ans.** The complications of tracheostomy are as follows:

- Bleeding at tracheostomy site
  - **Immediate bleeding** is due to poor hemostasis.

- Late bleeding is an ominous sign and suggests **trachea-innominate fistula**. The patient should be taken to operation theatre and immediate fiberoptic bronchoscopy and control of bleeding is to be done to save patient's life. Temporary bleeding control can be attained by finger compression of the site
- Blockage of tube can occur with blood clots and mucus
- Subcutaneous emphysema in neck
- Esophageal injury
- Injury to recurrent laryngeal nerve
- Air trapping in lungs can lead to pneumothorax and/or pneumomediastinum
- Infection at tracheostomy site or airway infection
- Tracheomalacia
- Tracheal stenosis
- Tracheoesophageal fistula
- Accidental tube removal.

### Prevention

- Appropriate size tube should be used for the procedure
- Good tracheostomy care
- Suctioning to remove clots and mucus plugs
- Regular checkup by ENT specialists
- Humidifying the air that passes through the tracheostomy tube
- The tracheostomy site should be routinely cleaned to prevent infection.

### Q73. Classify suture materials.

Ans.

The classification of suture materials is as follows:

Absorbable natural	<ul style="list-style-type: none"> <li>• Plain catgut</li> <li>• Chromic catgut</li> </ul>		
Absorbable synthetic	<ul style="list-style-type: none"> <li>• Monocryl (polyglyceproner)</li> <li>• Vicryl (polyglactin 910)</li> <li>• Vicryl rapid</li> <li>• Dexon (polyglycolic acid)</li> <li>• PDS (polydioxanone)</li> </ul>		
Non-absorbable natural	<ul style="list-style-type: none"> <li>• Silk</li> </ul>		
Non-absorbable synthetic	<ul style="list-style-type: none"> <li>• Prolene [polypropylene]</li> <li>• Nylon</li> <li>• Ethilon [Polyamide]</li> <li>• Polyester</li> </ul>		
<table border="1"> <tr> <td> <b>Monofilament</b>            Catgut, monocryl, PDS, prolene, ethilon         </td><td> <b>Polyfilament</b>            Vicryl, vicryl rapid, dexion, silk, polyester         </td></tr> </table>		<b>Monofilament</b> Catgut, monocryl, PDS, prolene, ethilon	<b>Polyfilament</b> Vicryl, vicryl rapid, dexion, silk, polyester
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**Q74. Write a note on options of pain management in surgery.**

**Ans.** Pain is a sensory and an emotional experience that is brought about by actual or potential tissue damage or described in terms of tissue damage.

- **Acute pain:** Pain duration < 1 month and pain that resolves within hours or at the most few days after the withdrawal of the stimulus or wound healing.
- **Chronic pain:** Pain that persists > 1 month beyond the expected time of healing/recovery.

**Types of pain**

- Nociceptive pain (muscle or viscera or skin and subcutaneous tissue origin)
- Neuropathic pain (nerve origin)
- Psychogenic pain (mental origin).

**Pain control methods in chronic painful conditions are as follows:**

- **Local medications such as anesthetic drugs, topical analgesia, topical steroids**
- **Nerve stimulation procedures such as acupuncture, transcutaneous electrical nerve stimulation (TENS)**
- **The pain management step ladder for oral and parenteral analgesics**
  - Simple analgesics: Aspirin and NSAIDs
  - Second step analgesics: Tricyclics, Pregabalin
  - Third step: Mild opioids such as tramadol
  - Final stand: morphine—oral, parenteral or epidural—continuous or patient controlled
- Management of phantom limb as described in the question on amputation is an example of pain management for chronic conditions and should be described here as an **example**.

**Pain management of malignant conditions**

- This also begins with the analgesic step ladder and proceeds to further neurolytic nonsurgical and surgical techniques
- These include techniques such as celiac ganglion block for pain due to pancreatic cancer, intrathecal neurolysis, anterolateral cordotomy as well as radiation therapy for pain relief (This is described in the question on functional neurosurgery in neurosurgery section)
- Other methods of pain relief involve hormones such as steroids and anti-pituitary drugs, psychotherapy, group therapy, physiotherapy and anticonvulsants can also be used for pain relief in these conditions.

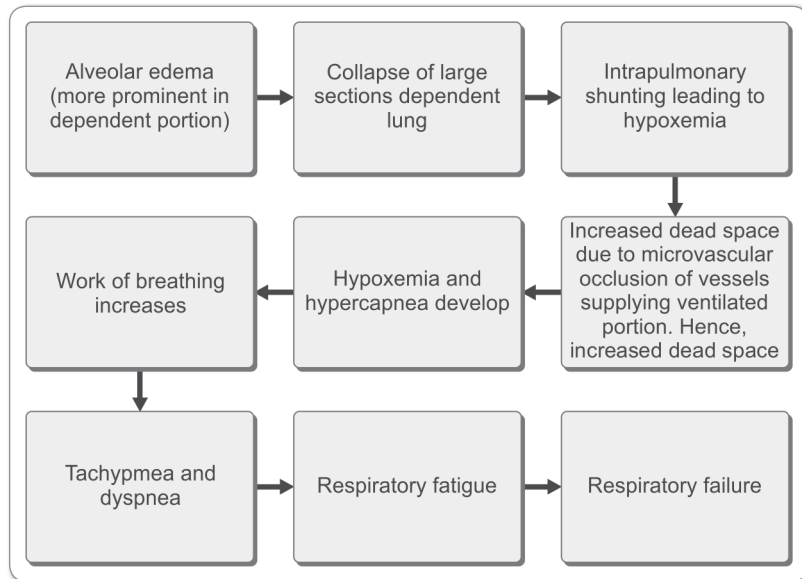
## MEDICINE IN SURGERY

**Q75. Write a note on ARDS.**

**Ans.** Acute respiratory distress syndrome is a clinical syndrome of severe dyspnea, hypoxemia, and diffuse pulmonary infiltrates leading to respiratory failure



- It is associated with common medical and surgical conditions including sepsis, severe trauma, multiple transfusions, pancreatitis, multiple transfusions, burns and drug overdose.
- Mortality : 26 to 44%. High index of suspicion and early identification is required for adequate management.
- Etiology
  - Pneumonias
  - Sepsis
  - Trauma
  - Post cardiopulmonary bypass
  - Near drowning
  - Toxic lung injury
- Diagnostic Criteria
  - Multiple definitions had been proposed till the one got published in 1994 proposed by AECC (American and European Consensus Conference) based on  $\text{PaO}_2/\text{FiO}_2$  ratio, PAWP and Chest Radiograph showing infiltrates.
  - Berlin Definition:
    - Within 1 week of known clinical insult or worsening respiratory symptoms
    - Bilateral opacities – not fully explained by effusions, lobar/lung collapse or nodules
    - Oxygenation
      - a. Mild –  $200 \text{ mm Hg} < \text{PaO}_2/\text{FiO}_2 \leq 300 \text{ mm Hg}$  with PEEP or CPAP  $\geq 5 \text{ cm H}_2\text{O}$
      - b. Moderate –  $100 \text{ mm Hg} < \text{PaO}_2/\text{FiO}_2 \leq 200 \text{ mm Hg}$  with PEEP or CPAP  $\geq 5 \text{ cm H}_2\text{O}$
      - c. Severe –  $\text{PaO}_2/\text{FiO}_2 \leq 100 \text{ mm Hg}$  with PEEP or CPAP  $\geq 5 \text{ cm H}_2\text{O}$
    - Origin of edema : Respiratory failure not fully explained by cardiac failure or fluid overload
  - ARDS can be multifactorial. Presence of two predisposing conditions can synergistically aggravate ARDS.
  - Predisposing factors: Old age, metabolic acidosis, alcohol consumption, decreased immunity.
- Pathophysiology/natural history
  - Exudative phase
    - Encompasses first 7 days
    - Injury to the cells (Type 1 pneumocytes) > endothelial injury > inflammatory exudation
    - Increased cytokines like interleukin 1, TNF > attracts leucocytes > activates leucocytes > further release of pro inflammatory chemokines
    - Cellular debris + protein aggregation = hyaline membranes
    - Endothelial injury of pulmonary vasculature causes microthrombi



- Proliferative phase
  - Usually lasts from 7th day to 21st day
  - Critical phase of mechanical ventilation ends in this stage
  - Tachypnea, dyspnea, hypoxemia still persist due to increased work of breathing
  - Histologically: Shift from neutrophilic to lymphocytic exudate and initiation of lung repair
  - Proliferation of type 2 pneumocytes
- Fibrotic phase
  - Most patients recover within 3 to 4 weeks after initial lung injury, some patients enter a protracted course with fibrotic phase.
  - Interstitial fibrosis and organization of the exudates ensues.
  - Acinar architecture is markedly disorganized.
  - Mortality substantial if clinical course prolongs to fibrotic phase.
- Treatment
  - General care
    - Treatment of the underlying cause
    - Fluid balance
    - Minimize procedures and their complications
    - Prompt identification of nosocomial infection and antibiotics
    - Adequate nutrition
  - Mechanical ventilation
    - Cornerstone of the management of ARDS

- ARDS specifically increase the risk of ventilator induced lung injury (VILI)
  - a. ARDS is a heterogeneous disorder with predisposition to dependent alveoli
  - b. Different areas with different pulmonary compliance are created
  - c. Mechanical ventilation causes overdistention of the more normal alveoli and hence leads aggravation of lung injury
  - d. Rhythmic collapse of the alveoli occurs during mechanical ventilation aggravating the lung injury
- Strategies to prevent VILI
  - a. Low tidal volume
    - » Low tidal volume, 6 mL/kg compared to conventional tidal volume 12 mL/kg improved survival and extubation rate among ARDS patients
  - b. High PEEP
    - » High PEEP prevents the collapse of alveoli at end expiration and hence prevents the extra lung injury occurring during the collapse
    - » Keeping PEEP high (around 12 to 15 cm H<sub>2</sub>O) prevents VILI and hence prolongs survival as compared to “normal” PEEP of 5 to 8 cm H<sub>2</sub>O
    - » Inversion of I : E ratio (inversion to expiration ratio) will prolong inspiration, leaves less time for expiration. Residual air present in the airways during expiration causes auto-PEEP and hence prevents collapse of alveoli.
  - c. Prone position
    - » Certain studies improved oxygenation in prone position due to recruitment of the basal alveoli but survival benefit was not obtained in them.
  - d. Other strategies
    - » High frequency ventilation (HFV) at 5–20 cycles/second and tidal volume 1–2 mL/kg
    - » Partial liquid ventilation (PLV) has revealed promising results initially but no survival benefit was seen
    - » ECMO—extracorporeal membrane oxygenation
    - » Data in support of the efficacy of “adjunctive” ventilator strategies are incomplete, hence should not be used routinely.
- Central pressure monitoring
  - Routine PCWP be monitored in these patients. Low normal PCWP should be maintained to prevent further dip in oxygenation
  - Diuretics and fluid restriction should be used in patients with concomitant cardiac dysfunction
- Glucocorticoids
  - Current evidences do not support routine treatment of ARDS with glucocorticoids.
- Functional recovery
  - Majority of the patients have normal functional recovery. Few patients with prolonged mechanical ventilation, ongoing pulmonary assault after development of ARDS undermine the functional recovery in a patient of ARDS.

**Q76. Write a note on diabetic ketoacidosis and hyperglycemic hyperosmolar state.****Ans.**

- DKA and HHS are acute metabolic complications of diabetes mellitus (DM)
- DKA was considered to be a hallmark of type 1 DM but it also occurs in individuals who lack immunologic features of type 1 DM and do not necessarily require insulin for control after the acute complication (DKA/HHS)
- Both of them represent a continuum of hyperglycemia with or without ketosis.

**DKA***Clinical features*

- Symptoms
  - Nausea/vomiting
  - Thirst/polyuria
  - Abdominal pain
  - Shortness of breath
- Physical findings
  - Tachycardia
  - Dehydration/hypotension
  - Tachypnoea/Kussmaul's breathing/respiratory distress
  - Fruity odor (acetone)
  - Abdominal tenderness (may resemble acute pancreatitis or surgical abdomen)
  - Lethargy/obtundation/cerebral edema/possibly coma
- Precipitating event
  - Inadequate insulin
  - Administration
  - Infection (pneumonia/UTI/gastroenteritis/sepsis)
  - Infarction (cerebral, coronary, mesenteric, peripheral)
  - Drugs (cocaine)
  - Pregnancy.

*Pathophysiology*

- Both deficiency of insulin and excess of glucagon are required for the development of DKA. Insulin deficiency promotes glycogenolysis, gluconeogenesis and lipolysis, all of which leads to the formation of acetyl-CoA and subsequently ketone bodies (acetoacetate, beta hydroxybutyrate and acetone)
- Insulin deficiency leads to inhibition of GLUT4 receptor, preventing the uptake of the glucose by the skeletal muscles and fat aggravating hyperglycemia
- Insulin deficiency shifts the balance of fatty acid metabolism from lipogenesis to lipolysis leading to production of large amount of fatty acids
- Normally increased fatty acids are converted into triglycerides in the presence of insulin, but insulin deficiency and hyperglucagonemia results into further degradation of fatty acids to acetyl-CoA, subsequently forming ketone bodies

- DKA is often precipitated physiological strain to the body leading to production of stress steroids, glucagon in a diabetic patient already deficient in insulin. DKA secondary to infections is common. Inadequate administration of insulin, non-compliance are important precipitating causes.

#### *Laboratory tests*

- Routinely monitored tests include complete hemogram, glucose, acid base gas analysis, blood urea, serum creatinine, Na, K, Cl, P
- Triad of features: Hyperglycemia, ketosis and metabolic acidosis
- Metabolic acidosis
  - Due to production of the ketone bodies
  - Arterial pH ranges from 6.9 to 7.3 based on the severity of the acidosis
  - $\text{HCO}_3^-$  are generally  $<10$  mEq/L. Hypocarbica is a feature of partial respiratory compensation
  - Anion gap is high in DKA. It is generally in the range of 20 to 40 mEq/L
- Hyperglycemia
  - It is usually in the range of 500 mg/dL
  - Causes of euglycemic DKA has been described below:
    - Obtunded patient with decreased oral intake
    - Pregnancy
    - Partially/poorly treated DKA with only insulin and no IV fluids
- Ketosis
  - Detected with urinary ketone dipsticks
  - It can be false negative in situations with excessive beta hydroxybutyrate (a ketone) which cannot be detected by nitroprusside test generally employed in the dipsticks
  - Serum beta-hydroxybutyrate levels are required for further diagnosis
- Potassium is actually deficient in the body, however, normal or hyperkalemia are not uncommon in DKA (secondary to acidosis or decreased renal clearance as in AKI)
- Sodium is low in measurement as a consequence of the hyperglycemia
  - Corrected sodium :  $\text{measured sodium} + 0.016 (\text{glucose} - 100)$
- Hypertriglyceridemia is seen in certain patients
- Leucocytosis and hemoconcentration can be present due to underlying infection and severe dehydration
- Serum amylase should be evaluated to differentiate from pancreatitis, a common condition presenting with abdominal pain
- Serum osmolarity =  $(2 \text{ sodium}) + (\text{BUN}/2.8) + (\text{Glucose}/18)$ 
  - Mildly or moderately increase in DKA (300–320 mOsm/L)
  - Very high in HHS (330–380 mOsm/L)

#### *Differential diagnosis*

- Lactic Acidosis
  - Decreased tissue oxygen delivery in settings of hypotension, shock and dehydration
  - Serum lactate  $> 5$  mmol/L normal ketones and normal glucose in pure lactic acidosis
  - Can be a contributing factor in DKA with sepsis or dehydration.

- Starvation ketosis
  - Normal blood glucose
  - High urinary ketones and normal blood ketones
  - Arterial pH is normal mildly raised anion gap.
- Alcoholic ketoacidosis
  - Normal pH with respiratory alkalosis often present due to delirium tremens, agitation
  - Blood ketones and urinary ketones invariably raised
  - Normal blood glucose or hypoglycemia
  - Treated with IV fluids, thiamine and carbohydrates.
- Uremic acidosis
  - BUN, *S. creatinine* are raised
  - Normoglycemia.
- Toxic ingestions
  - Proper history and examination can rule out salicylate and ethylene glycol, common toxic ingestions with high anion gap and metabolic acidosis.

### *Treatment*

After initiating IV fluid replacement and insulin therapy, the agent or event that precipitated the episode of DKA should be sought and aggressively treated. If the patient is vomiting or has altered mental status, a nasogastric tube should be inserted to prevent aspiration

- **Fluids**
  - Fluid deficit of the order of 5 to 10 L are common in DKA and even larger in HHS.
    - Water deficit in liters:  $0.6 \text{ Wt. in kgs (sodium/140 - 1)}$
  - Initial bolus of 0.9% Saline: 2 to 3 L over 1 to 3 hours
  - Meantime, the laboratory investigations are available, further fluid management is based on the calculation of water deficit.
  - 0.45% saline: One-half of the total body deficit + urinary loss to be corrected in next 12 hours and remaining over next 24 hours.
  - Use of 0.45% saline reduces the incidence of hyperchloremic metabolic acidosis seen due to rapid volume expansion
  - When plasma glucose reaches 250 mg/dL: change to 0.45% saline + 5% Dextrose saline to maintain the blood glucose in level of 200 to 250 mg/dL for next 24 hours along with insulin infusion to allow slow equilibrium of osmotically active substances across cell membrane
  - Rapid fluid replenishment in hyperosmolar patients can precipitate cerebral edema and should be avoided
- **Insulin**
  - I.V. bolus of 0.1 IU/kg immediately followed by I.V. infusion of 0.1 IU/kg/hr to provide continuous circulating insulin
  - Insulin should not be administered if S. Potassium is less than 3.3 mEq/L
  - If glucose concentration doesn't decrease by 50 mg/dL within 1st hr, second bolus of 0.1 IU/kg should be given and infusion rate increased by 50% or 100%
  - Insulin rate should be decreased to 0.05 to 0.1 IU/kg/hr

- As soon as the patient becomes orally accepting, long acting insulin + short acting insulin should be administered. Insulin infusion should be stopped after 30 minute of subcutaneous insulin administration to prevent decrease in S. insulin concentration
- A brief period of insulin deficit during transition can cause relapse.
- **Potassium**
  - DKA is a condition with potassium deficit. (3 to 5 mEq/kg)
  - Potassium should not be administered before laboratory investigations/ECG are available
  - When ECG features of hyperkalemia (tall T waves, QRS widening) are absent or S.  $K^+$  is  $<5.5$  mEq/L, potassium should be administered at 10 mEq/hr
  - When S.  $K^+ < 3.5$  mEq/L, administer 40 to 80 mEq/hr
  - Causes of hypokalemia in DKA therapy
    - Insulin mediated intracellular shift
    - Correction of acidosis causing intracellular shift
    - IV fluids causing brisk diuresis with urinary potassium loss
- **Bicarbonate**
  - Despite bicarbonate deficit, it need not to be administered. Insulin causes metabolic conversion of ketones to bicarbonate
  - No benefit of bicarbonate therapy in clinical studies of DKA patients
  - Bicarbonate should be administered when
    - pH is  $< 6.9$ , hemodynamic instability with pH  $< 7.1$
    - with hyperkalemia on ECG findings
  - Given as : 50 mEq/L in 200 mL sterile water with 10 mEq/L of KCl per hour until pH  $> 7.0$
  - Potassium is given to prevent acute drop in  $K^+$  which occurs with correction of acidosis.
- **Phosphate**
  - No benefit of phosphate administration.

#### *Monitoring*

- Frequent laboratory investigations, fluid input/output charting and vital signs are the cornerstone of the management of DKA. The importance of a comprehensive chart of clinical and laboratory parameters as a function of insulin cannot be underestimated
- Bladder catheterization is required if patient cannot void at will
- ICU and continuous ECG monitoring are required if pH is less than 7.3
- Baseline: Complete hemogram, BUN, creatinine, electrolytes, glucose, ketones, lactate, urinalysis, ABG, ECG and upright chest radiograph
- Hourly: Glucose and electrolytes till IV insulin is given; then 2–4 hourly
- 6 hourly: BUN, creatinine, ketones
- Intensive monitoring is generally required for the first 12 hours.

Goal is to correct DKA with its metabolic complications completely within 36 hours.

#### **Q77. Write a note on adrenal insufficiency and its management.**

##### **Ans. Etiology**

- **Primary (Adrenal):** Deficiency of the secretion of the glucocorticoids and mineralocorticoids from the adrenal glands

- Autoimmune adrenalitis
- Autoimmune polyglandular syndrome 1 and 2 (APS1 and APS2)
- Congenital adrenal hyperplasia
- X-linked adrenoleukodystrophy
- Infections: Tubercular, HIV, CMV
- Infiltrations: Lymphomas, sarcoidosis, amyloidosis, hemochromatosis
- Ketoconazole, suramin, trilostane
- **Secondary:** Inappropriately low stimulus from the pituitary due to low secretion of the ACTH.
  - Pituitary tumors (endocrinally active or inactive)
  - Intracranial SOLs: Meningioma, craniopharyngioma, ependymoma
  - Autoimmune hypophysitis
  - Sheehan syndrome
  - Pituitary irradiation
- **Tertiary:** Hypothalamic signal disruption/suppression to pituitary for the secretion of ACTH.
  - Chronic glucocorticoid excess followed by sudden withdrawal (most common).

### Clinical Manifestations

- Primary adrenal insufficiency commonly presents with the symptoms and signs of mineralocorticoid as well as glucocorticoid deficiency while secondary adrenal insufficiency presents with just glucocorticoid deficiency with history of prolonged steroid ingestion followed by abrupt stopping of the drug or intracranial lesion
- Adrenal androgen secretion is disrupted in both primary and secondary adrenal insufficiency
- Chronic adrenal sufficiency
  - Lethargy, fatigue, loss of energy, anorexia
  - Myalgia, joint pain
  - Pigmentation is the differentiating feature between primary and secondary adrenal insufficiency.
    - Primary: Decreased glucocorticoids >> raised ACTH >> raised POMC (Pro opiomelanocortin) >> increased pigmentation
    - Secondary: Absent or abnormal ACTH and POMC >> paleness
  - Hyponatremia
  - Hypoglycemia
  - Hyperkalemia
  - Hypotension, postural hypotension
  - Fluid loss due to reduced mineralocorticoids can cause AKI
- Acute adrenal insufficiency
  - Non specific complain of lethargy and weakness
  - Postural hypotension progressing to hypovolemic shock
  - Acute abdomen can be presenting feature of acute adrenal insufficiency
  - Nausea, vomiting



- Predisposing factors to acute insufficiency in case of primary chronic insufficiency
  - Infections
  - Hyperthyroidism
  - Surgical stress.

### Treatment

- Acute adrenal insufficiency requires immediate fluid therapy and glucocorticoid administration
- Diagnosis should not hamper the early initiation of the treatment. Random S. cortisol level sample should be withdrawn and glucocorticoid (hydrocortisone 100 mg stat f/b 100 to 200 mg/ day in divided doses) should be administered. Cosyntropin test and other definitive diagnostic techniques are to be deferred until a later time
- Mineralocorticoid, are not to be given separately until hydrocortisone dose is > 50 mg/ day because the daily requirement is met by intrinsic activity of hydrocortisone
- Glucocorticoid replacement
  - Prolonged glucocorticoid administration is the cornerstone of the treatment of acute adrenal insufficiency
  - Daily requirement: 15 to 20 mg of hydrocortisone in two –three divided doses
  - Dose equivalence  
 $1 \text{ mg hydrocortisone} = 0.25 \text{ mg prednisolone} = 0.025 \text{ mg dexamethasone}$
  - Half of the dose should be administered in the morning. However, present delivery systems and preparations do not mimic the normal physiological secretion of the body
  - Long acting preparations like dexamethasone and prednisolone are not preferred because of prolonged stimulation of the glucocorticoid receptor causing disruption of the normal cycling secretion
  - Monitoring
    - Signs, symptoms, blood pressure and volume status monitoring
    - In primary adrenal insufficiency, monitoring of thyroid status for autoimmune thyroiditis
    - If daily dose of glucocorticoid > 30 mg/day of hydrocortisone, then a bone marrow density evaluation is required
  - Dose modification is required in conditions of stress. Dose should be doubled by the patient in fever, while undergoing operation, vomiting and trauma
- Mineralocorticoid replacement
  - Mineralocorticoid supplementation should begun at a dose of 100 to 150  $\mu\text{gm/day}$
  - Monitoring is done by blood pressure and postural hypotension evaluation
  - 40 mg of hydrocortisone is equivalent to 100  $\mu\text{gm}$  of fludrocortisone
  - Dose is doubled, i.e. 200  $\mu\text{gm/day}$  in summers in tropical countries
- Adrenal androgen replacement
  - It is optional, indicated in patients with lethargy and weakness after optimum dose of glucocorticoid and mineralocorticoid replacement

- 25 to 50 mg of DHEA once a day
- It is also indicated in females with features of adrenal insufficiency.

**Q78. Write a note on myxedema coma.**

**Ans.**

- Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia and other symptoms related to slowing of functions in multiple organs
- It is a medical emergency with a high mortality rate
- Early recognition and therapy of myxedema coma are essential. Treatment should begun on the basis of clinical suspicion without waiting for laboratory results
- A history obtained from family members often reveals antecedent symptoms of thyroid dysfunction followed by progressive lethargy, stupor and coma.

**Clinical presentation**

- The function of virtually every organ system and the activity of many metabolic pathways are slowed in severe hypothyroidism
- Hallmarks of myxedema coma are:
  - Decreased mental status and
  - Hypothermia
- Hypotension, bradycardia, hyponatremia, hypoglycemia and hypoventilation are often present as well. Puffiness of the hands and face a thickened nose, swollen lips and an enlarged tongue may occur secondary to nonpitting edema with abnormal deposits of mucin in the skin and other tissues (myxedema).

*Neurologic manifestations*

- Despite the name, myxedema coma patients do not necessarily present in coma, but do manifest lesser degrees of altered consciousness
  - May present in the form of confusion with lethargy and obtundation
  - More activated presentation may occur with prominent psychotic features, so-called myxedema madness
  - Untreated patients will progress to coma
- It has been reported that focal or generalized seizures may occur, sometimes due to concomitant hyponatremia, and status epilepticus.

*Hyponatremia*

- Hyponatremia is present in approximately one-half of patients with myxedema coma
- It can be severe and may contribute to the decrease in mental status. Cause is uninterrupted excess secretion of ADH (normally under negative inhibition of T4 hormone).

*Hypothermia*

- Hypothermia is present in many patients with myxedema coma. Temperature as low as 23°C has been seen with myxedema coma. It is due to the decrease in thermogenesis
- The severity of hypothermia is related to mortality in severe hypothyroidism.

*Hypoventilation*

- Spectrum of hypoventilation from snoring to cheyne Stokes breathing to hypercapnia to respiratory arrest may be seen with myxedema coma
- Causes
  - Decreased respiratory drive
  - Muscle weakness
  - Mechanical obstruction by a large tongue and
  - Sleep apnea (due to associated obesity)
- Some patients require mechanical ventilation.

*Hypoglycemia*

- Hypoglycemia is commonly seen in hypothyroidism
- Causes:
  - Decreased oral intake
  - Decreased gluconeogenesis due to metabolic slowdown
  - Infection, sepsis causing increased uptake
  - Concomitant adrenal insufficiency.

*Cardiovascular abnormalities*

- Hypothyroid patients have diastolic hypertension, even though cardiac output is reduced and pulse pressure gets narrowed.
- Severe hypothyroidism is associated with bradycardia, decreased myocardial contractility, a low cardiac output and sometimes hypotension
- Pericardial effusion may be present
  - Clinical manifestations: diminished heart sounds, low voltage on electrocardiogram, and a large cardiac silhouette on chest x-ray and raised JVP
  - Cardiac tamponade is rare but not unheard in myxedema coma
- All of the cardiac abnormalities are reversible.

**Diagnosis**

Cornerstone of diagnosis is high index of suspicion

- The diagnosis of myxedema coma is initially based upon the history, physical examination, and exclusion of other causes of coma
- Previous history of thyroid surgery, radiation treatment and drug therapy for hypothyroidism should always be asked in a patient of suspected myxedema coma
- Myxedema coma is differential in an elderly patient with coma or depressed mental status who also has hypothermia, hyponatremia, and/or hypercapnia
- Before therapy, glucocorticoid with a thyroid hormone is given. Blood should be drawn for measurements of serum thyrotropin (TSH), free T4 and cortisol
  - High serum TSH and low free T4 values : primary hypothyroidism
  - Low serum TSH with a low free T4 value: Secondary to hypothalamic or pituitary dysfunction
- Treatment should be instituted in patients with presumed myxedema coma without waiting for laboratory confirmation.

**Treatment**

- Myxedema coma is an endocrine emergency and should be treated aggressively
- The mortality rate remains high at 30 to 40 percent
- Risk factors :elderly patients and those with cardiac complications, reduced consciousness, persistent hypothermia and sepsis
- Treatment consists of thyroid hormone, supportive measures and appropriate management of coexisting problems such as infection and adrenal insufficiency.

*Thyroid hormone*

- The optimal mode of thyroid hormone therapy in patients with myxedema coma is controversial
- No clinical trials comparing the efficacy of different treatment regimens
- Risk of precipitating myocardial infarction or atrial arrhythmias with high doses of T3 or T4 hormones
- This risk must be accepted because of the high mortality of untreated myxedema coma
- In one randomized trial of 11 patients, those who received a 500 µg loading dose of T4, followed by 100 µg daily, had a lower mortality than those treated with 100 µg daily without a loading dose
- Whether patients with myxedema coma should be treated with T4, T3 or both is controversial. Some experts favor administration of T3, while others favor T4 only while another school of thoughts prefer a combination of T4 and T3
- Combination is theoretically preferred
  - Activity of triiodothyronine (T3) is greater and its onset of action is more rapid than T4
  - Conversion of T4 to T3 is impaired due to both hypothyroidism and any concurrent illness
- T4 should be given intravenously when available because gastrointestinal absorption may be impaired
  - Loading dose of 500 µg
  - Daily dose of 100 µg/day (1.6 µg/kg) intravenously initially and orally when feasible.
- T3 may be given simultaneously
  - Loading dose of 5 to 20 µg followed by
  - 2.5 to 10 µg every eight hours
  - T3 is continued until there is clinical improvement and the patient is stable.

*Glucocorticoids*

- Patients with secondary hypothyroidism may have associated hypopituitarism and secondary adrenal insufficiency
- Until the possibility of coexisting adrenal insufficiency has been excluded, the patient must be treated with glucocorticoids in stress doses (e.g. 100 mg hydrocortisone given intravenously after every shows).

**Supportive measures**

- Myxedema coma is an endocrine emergency. Requires intensive care unit for the management

- Mechanical ventilation may be required. Acid base and  $\text{PCO}_2$  monitoring is necessary till consciousness improves
- Intravenous fluids
  - Dilute fluids should be avoided in hyponatremic patients
  - CVP monitoring might be required in patients with cardiac or renal comorbidities
- Hypotension
  - Volume depletion has to be corrected, if present
  - Thyroid hormone therapy improves cardiac output over a period of hours to days alleviating hypotension
  - Vasopressors might need to be given until T3 and T4 show metabolic improvement
- Hypothermia
  - Indicated only if temperature  $< 30^\circ\text{C}$
  - Passive rewarming with a blanket is preferred
  - Active rewarming carries a risk of vasodilatation and worsening hypotension
- As with any critically ill, comatose patient, empiric administration of antibiotics should be considered until appropriate cultures are proven negative.

**Q79. Write a note on thyroid storm.**

**Ans.**

- A rare, life-threatening condition of thyrotoxicosis
- often precipitated by an acute event such as thyroid or nonthyroidal surgery, trauma, infection, an acute iodine load, or parturition
- The advent of appropriate preoperative preparation of patients undergoing thyroidectomy for hyperthyroidism has led to a dramatic reduction in the prevalence of surgically-induced thyroid storm
- Total T4 and T3 levels might be similar to those seen in uncomplicated patients, the free T4 and free T3 concentrations were higher in patients with thyroid storm.

**Diagnosis**

- Based upon clinical findings. Patients with severe and life-threatening have an exaggeration of the usual symptoms of hyperthyroidism
- Symptoms include:
  - Tachycardia to rates that can exceed 140 beats/minute
  - Congestive heart failure
  - Hypotension
  - Cardiac arrhythmia
  - Hyperpyrexia to  $104$  to  $106^\circ\text{F}$  is common
  - Agitation, anxiety, delirium, psychosis, stupor or coma are also common and are considered by many to be essential to the diagnosis
  - Severe nausea, vomiting, diarrhea, abdominal pain, or hepatic failure with jaundice can also occur
- No universally accepted criteria or validated clinical tools for diagnosing thyroid storm

- In 1993, Burch and Wartofsky introduced a scoring system using precise clinical criteria for the identification of thyroid storm. Many other criterias were also proposed but none of them is specific enough to be accepted in clinical practice
- Thyroid function tests (TSH, free T4 and T3) should be assessed in all patients in whom there is a clinical suspicion of thyroid storm
- Degree of hyperthyroidism is not a criteria for the diagnosis of thyroid storm
- Other nonspecific laboratory findings:
  - Hyperglycemia due to release of catecholamines
  - Mild hypercalcemia due to hemoconcentration and increased bone resorption.

**Treatment**

- Early therapeutic intervention is important due to high incidence of complications
- The therapeutic options for thyroid storm are the same as those for uncomplicated hyperthyroidism except that the drugs are given in higher doses and more frequently
- Supportive therapy and recognition and treatment of any precipitating factors (e.g. infection), in addition to specific therapy directed against the thyroid is vital to decrease the mortality.
  - Management protocol differs in patient according to their clinical status
  - Some may require fluid replenishing due to overt sepsis and hypovolemia
  - While many patients presenting with heart failure may require diuresis
  - Metabolism of hepatic metabolized drugs is enhanced. This might lead to therapeutic failure of antihypertensive and antiepileptic medications
- Antithyroid medications:
  - The therapeutic regimen typically consists of multiple medications, each of which has a different mechanism of action
  - Beta blocker propranolol in a dose to achieve adequate control of heart rate, typically 60 to 80 mg orally every four to six hours
    - Prevent catecholaminergic reactions
    - Non selective beta blocker propranolol prevent conversion of T4 to T3 in peripheral tissues and hence, it is preferred in thyroid storm
    - Intravenous therapy is preferable in obtunded and severe thyroid storm patients. Regular blood pressure monitoring is vital to prevent hypotension during IV therapy
    - Contraindicated in patients with severe asthma or failure. Diltiazem is used for heart control in patients with airway disease
  - Propylthiouracil vs methimazole
    - Methimazole and propylthioural prevents formation of new hormone but are ineffective on preformed thyroid hormone stored in the glands
    - PTU (propylthiouracil) is short and fast acting and prevents peripheral conversion of T4 to T3 in addition to inhibiting synthesis while methimazole is longer acting, require daily dosing and provides a better control in hyperthyroidism as compared to propylthiouracil and is less hepatotoxic

- Propylthiouracil is used in rapid control in the ICU followed by gradual shifting of methimazole/carbimazole. Some advocate direct use of methimazole in less severe presentation of thyroid storm.
- Iodine Lugol's solution—10 drops every eight hours
  - Large doses of inorganic iodine prevents formation of new organic iodine compounds known as Wolf Chaikoff effect
  - The administration of iodine should be delayed for at least one hour after thionamide (PTU or methimazole) administration to prevent the iodine from being used as substrate for new hormone synthesis
- Glucocorticoids (hydrocortisone, 100 mg intravenously every eight hours)
  - Reduce T4 to T3 conversion
  - May have a direct effect on the underlying autoimmune process if the thyroid storm is due to Graves' disease and treat potentially associated limited adrenal reserve.

# SECTION

# 2

## **Gastrointestinal, Hepatobiliary and Pancreatic Surgery**

- Esophagus and Diaphragm
- Stomach and Duodenum
- Small Intestine
- Large Intestine
- Appendix
- Rectum and Anal Canal
- Liver
- Gallbladder and Bile Duct
- Pancreas
- Spleen
- Hernias
- Gastrointestinal Oncosurgery
- Miscellaneous (Burst Abdomen, Peritonitis, Mesenteric Cyst)





## ESOPHAGUS AND DIAPHRAGM

**Q1. Write a note on diaphragmatic hernias.**

**Write a note on congenital diaphragmatic hernia.**

**Write a note on hiatus hernia (Acquired diaphragmatic hernia).**

**Discuss paraesophageal hernia.**

**Write a note on sliding hernia.**

**(Imp: Always Remember:**

1. There are two primary types of diaphragmatic hernias—congenital and acquired.
  2. There are two sliding hernias—diaphragmatic sliding and inguinal sliding.
- SO, read the questions carefully.)

**Ans.** The diaphragmatic hernias can be congenital (Bochdalek and Morgagni) or can be acquired and should be described as follows:

### **Congenital diaphragmatic hernia**

- Also called **Bochdalek hernia** or posterolateral hernia which is more common than the other congenital diaphragmatic hernia called **Morgagni's hernia** (right anteromedial/presternal hernia/Larrey's hernia with transverse colon as the most common content).

### **Mnemonic: BPL (Bochdalek, posterior, left) and RAM (right, anterior, Morgagni)**

- Incidence—1 in 5000 live births
- Most common site—left posterolateral
- It is rarely bilateral.

### *Etiology*

- Failure of normal closure of the pleuroperitoneal canal
- As a result, abdominal contents herniate through the resultant defect which leads to the pathophysiology of the disease.

### *Pathophysiology*

- Compression of the lung causes pulmonary hypoplasia which is bilateral.
- Ipsilateral lung is more affected than the contralateral lung.
- Also causes pulmonary hypertension due to increase in the medial muscular thickness of the artery walls.

### *Clinical features*

- Presentation is just after birth or diagnosed by prenatal ultrasound.
- Respiratory distress + dextrocardia + scaphoid abdomen is the classic triad of findings.
- There is dyspnea, cyanosis and infant also has chest retractions.

### *Investigations*

- Diagnoses can be established in the prenatal ultrasound.
- Postnatal chest X-ray—gastric air bubble in chest or bowel loops in the chest.
- Pneumothorax can occur on the opposite side of the CDH.

*Treatment*

- When diagnosed **prenatally** – open fetal surgery or fetal tracheal occlusion is being evaluated as a treatment option (In this, fetus is kept attached to the umbilical cord for around half an hour to allow surgeon to perform Ex-utero fetal surgery for various conditions)
- Postnatally, the first priority is the stabilization of the respiratory symptoms
- Early surgery without respiratory stabilization adds to the physiologic compromise and is not recommended.

*Initial management*

- Gentle ventilation with permissive hypercapnia and stable hypoxemia is a better management option associated with a higher survival rate (75%) for these infants.
- If the child has an indication for ECLS (extracorporeal life support), ECLS should be initiated and CDH repair should be done after the completion of ECLS therapy. ECLS is giving respiratory and/or cardiac support outside body to a newborn when his own organs cannot fulfil the requirements.

Once the respiratory condition is taken care of by above measures, the infant is operated of 1 to 3 days.

*Operative steps of importance*

- Viscera are reduced into the abdominal cavity.
- Hernia sac if present is excised.
- Defect closed primarily or using a mesh. The aim is a tension free repair.

*Prognosis*

- Most important prognostic factor is pulmonary hypoplasia
- Pulmonary hypertension
- Risk of bleeding and other complications with ECMO is more common in infant <2.2kg and <35 weeks age, again associated with a poor prognosis
- Other poor prognostic factors include:
  - Extradaphragmatic anomalies
  - Need for ECMO
  - Polyhydroamnios
  - Size of defect.

**Hiatus hernia or acquired diaphragmatic hernia***Types***1. Sliding hernia**

- Overall most common type
- There is an upward displacement of gastroesophageal junction into the posterior mediastinum
- Seen to occur more often with short esophagus and patients with inguinal hernia
- Size correlates with the severity of symptoms.

**2. Rolling (true paraesophageal hernia)**

- PEH is more common in females

- Here, the gastroesophageal junction is normally positioned with upward herniation of stomach anterior to esophagus as posteriorly GE junction is fixed normally.
- 3. **Mixed variety (Type 1 and 2)**
  - M.c. of the paraesophageal variety
  - Starts as a sliding hernia and progresses to include fundus and body of stomach
- 4. **Paraesophageal hernia containing other intra-abdominal organs**
  - Most serious of all the hiatal hernias
  - Sac contains other organs such as spleen, colon or small intestine.

#### *Clinical features*

- Heart burns
- Epigastric and chest pain, dysphagia
- Regurgitation
- Postprandial fullness
- Hematemesis and anemia are also seen with the paraesophageal variety.

#### *Investigations*

- Barium swallow—the most important diagnostic test
- Endoscopy—mucosal erosions can be found
- Manometry—motility disorder if co-existent can be identified.

#### *Complications*

- **Microscopic complication** – esophagitis
- **Cameron ulcer/riding ulcer** – occurs due to the constant abrasive force on the stomach as it rubs against the diaphragmatic hiatus up and down with respiration.

#### *Management*

- Medical management with acid reducing agents followed by surgery
- **Indications of primary surgery**
  - All paraesophageal hernias
  - Hernias with narrow opening with interruption of barium flow to stomach
- Principles of surgery
  - Excision of sac
  - **Mesh to be placed if the defect is > 8 cm**
  - Cruroplasty (diaphragmatic crura are sutured together to prevent migration of the GE junction into the thorax and resultant postoperative GERD) to be done
- **Approach** – transabdominal (open or laparoscopic) is preferred over transthoracic approach.

#### **Sliding inguinal hernia (Hernia-en-glissade)**

- When part of the wall of the sac (usually the posterior wall) is formed by a viscus, the hernia is called a sliding hernia
- Incidence—2 to 5% of all indirect inguinal hernias.

#### *Pathophysiology*

- Advancing age, increased intra-abdominal pressure and increasing obesity are risk factors as they lead to laxity of tissues

- As the retroperitoneal connective tissues become infiltrated with fat and as the muscles and fasciae become weaker with age the posterior parietal peritoneum advances through the ring
- Also, a loop of bowel and even the medial leaf of the mesentery may slide through the ring and form the posterior wall of the hernial sac.

### Organs

- **Right side**—Cecum, the ascending colon, the appendix, the appendiceal mesentery, the terminal ileum and the mesentery of the terminal ileum may form the posterior wall of the sac
- **Left side**—Sigmoid colon
- **Both sides**—Ovary, fallopian tubes, urinary bladder can also form the posterior wall.

### Clinical features

- Most common in aged males on left > right
- Hernias are of long duration, usually reducible and low incidence of intestinal obstruction.

### Diagnoses

- A large hernia of long standing in an elderly patient should cause suspicion of a sliding hernia
- Barium enema may reveal colon outside of the abdominal cavity
- The diagnosis will most often be made at the operating table.

### Treatment

- Accurate diagnosis of a sliding indirect inguinal hernia usually is not made until the sac is opened
- For this reason, it is important to open all indirect inguinal hernial sacs on the anterior surface so as to avoid opening into bowel which might be forming the posterior wall of a sliding hernia.

**Aim of surgery**—Peritonealize the extraperitoneal herniated organs so as to prevent recurrence.

**Two approaches**—Intra-abdominal (LaRoque) or inguinal – Zimmerman and Laufman. The LaRoque technique is the preferred technique.

- **The LaRoque technique (intra-abdominal approach)** in which the peritoneal cavity is entered above the internal ring allows accurate definition of the pathological anatomy and effective repair of the hernia. It should be used in all true sliding indirect inguinal hernias.
- **Inguinal approach** - Zimmerman and Laufman described placing a purse-string suture about the neck of the sac extending as high on the anterior surface as possible and on the posterior side as close as is safe to the reflection of the peritoneum on the colon. As the purse-string is pulled together the bowel is turned upward and the sac is closed. The sac and bowel are reduced into the abdominal cavity. The transversalis fascia then is closed snugly about the cord to reconstruct the internal ring.

**Q2. Define portal hypertension and enumerate the causes of portal hypertension. Define portal hypertension and describe the anatomy and pathophysiology of portal hypertension.**

**Ans.** Portal vein is formed by the union of superior mesenteric vein and splenic vein behind the neck of the pancreas.

### Definition

- Portal pressure higher than 5 mmHg or 8 cm water
- 8 to 10 mmHg pressure is required for portosystemic collateralization and > 12 mmHg is required for actual variceal bleeding.

### Anatomy of portosystemic circulation

The important sites of portosystemic collaterals are:

- At the lower end of oesophagus**
  - Oesophageal tributaries of left gastric vein (portal)
  - Oesophageal tributaries of hemiazygos vein (systemic)
- Around the umbilicus**
  - Para umbilical vein (portal)
  - Superior and inferior epigastric veins (systemic)
- Lower rectum and anal canal**
  - Superior rectal vein (portal)
  - Middle and inferior rectal veins (systemic)
- Retroperitoneum**
  - Tributaries of superior and inferior mesentric veins (portal)
  - Posterior abdominal and subdiaphragmatic veins (systemic)
- At the back of the colon**
  - Right and left colic veins (portal)
  - Right and left renal veins (systemic)

### Pathophysiology

Increased portal venous resistance is the main etiology.

### Causes

Prehepatic	Intrahepatic	Posthepatic
Splenic vein thrombosis (m.c. cause is acute pancreatitis)	<b>Presinusoidal</b> <ul style="list-style-type: none"> <li>• Schistosomiasis (m.c. presinusoidal cause)</li> <li>• Myeloproliferative disease</li> <li>• Focal nodular hyperplasia</li> <li>• Chronic viral hepatitis</li> <li>• Amyloidosis</li> <li>• Wilson disease</li> <li>• Hemochromatosis</li> <li>• Sarcoidosis</li> <li>• Metabolic storage diseases</li> </ul>	Budd Chiari syndrome (Hepatic vein thrombosis)
Portal vein thrombosis (m.c. prehepatic cause)		Constrictive pericarditis
Cavernous transformation of the portal vein	<b>Sinusoidal</b> <ul style="list-style-type: none"> <li>• Cirrhosis [m.c. cause overall]</li> <li>• Acute fatty liver of pregnancy</li> </ul>	IVC web

Contd...

Contd...

Prehepatic	Intrahepatic	Posthepatic
Splenic AV fistula		Chronic right ventricular failure
Tropical splenomegaly	<b>Postsinusoidal</b> <ul style="list-style-type: none"> <li>• Veno-occlusive disease</li> </ul>	Tricuspid insufficiency

**Factors leading to portal hypertension**

- Increased passive resistance secondary to fibrosis and regenerative nodules
- Increased hepatic vascular resistance caused by norepinephrine, endothelin and other humoral vasoconstrictors
- Increased portal venous inflow secondary to a hyperdynamic systemic circulation and splanchnic hyperemia.

**Q. Enumerate the various clinical presentations/sequelae of a patient with portal hypertension.****Ans.**

Splenomegaly	<ul style="list-style-type: none"> <li>• Most common clinical finding</li> <li>• Symptomatic splenomegaly can manifest as               <ul style="list-style-type: none"> <li>– Early satiety</li> <li>– Upper abdominal heaviness</li> <li>– Diarrhea</li> <li>– Poor gastric emptying</li> </ul> </li> </ul>
Esophageal varices	<ul style="list-style-type: none"> <li>• 30% of patients with compensated cirrhoses and 60 % of patients with decompensated cirrhoses have varices</li> <li>• 30% of these bleed sometime in their life</li> </ul>
Ascites (can be intractable)	<ul style="list-style-type: none"> <li>• Hypoalbuminemia</li> <li>• Increased lymphatic transudation from liver</li> <li>• Salt and water retention are suggested etiologies</li> </ul>
Bowel wall congestion and edema	<ul style="list-style-type: none"> <li>• Dyspepsia</li> <li>• Malabsorption</li> <li>• Anorexia</li> </ul>

**Q. Explain the management of acute esophageal variceal hemorrhage.****Write a note on esophageal varices.****Write a note on TIPS.****Ans. Variceal Hemorrhage**

Important relations with cirrhoses

- 30% of patients with compensated cirrhoses and 60 % of patients with decompensated cirrhoses have varices.
- 30% of these bleed sometime in their life.
- 1/3 of those who bleed die.
- Of the remaining, upto 70% have a re-bleeding episode.
- Risk of bleeding is highest in the first few days and decreases thereafter.

**Management steps of bleeding esophageal varices**

1. **Resuscitation** with fluids, electrolyte correction, acidosis correction.
2. **Rule out coagulopathy** (Vitamin K, platelet, FFP) and prevent encephalopathy (Lactulose/neomycin).
3. **Pharmacotherapy**

**Vasopressin**

- Mechanism—Vasoconstriction of the splanchnic circulation
- Dose—0.2 unit/kg wt, dissolved in 200 ml of 5% dextrose, over 20 minutes.
- **Disadvantages**
  - Colicky abdominal pains and diarrhea
  - Angina pains, so it is contraindicated in the elderly
  - Produce temporary control of bleeding in about 80% of cases.

**Somatostatin/octreotide**

- Lower the intravariceal pressure without significant side effects
- Dose : bolus 50 mg followed by continuous infusion of 50 mg/hr for 24 hrs.

**Beta blockers**

- Mechanism—decrease cardiac output and systemic hyperdynamic circulation
- Dose 20 to 60 mg bid
- Reduces 40% of bleeding episodes
- Does not reduce mortality once the bleed has occurred.

**Antibiotics**

- Should be started to prevent sepsis due to impaired gut barrier function and bacterial translocation from the gut
- These are continued till at least 7 days along with the above drugs.

**4. Endoscopic Treatment (Sclerotherapy or Variceal Ligation)****- Sclerotherapy**

- Intra-variceal and/or para-variceal injection of 1–3 ml sclerosant usually ethanolamine oleate is given to occlude the venous channels
- Multiple sessions are required (2 weekly)
- Control bleeding in 80–95%
- About 50% rebleed

**Local complications (incidence – 30%)**

- Ulceration
- Stricture
- Perforation
- Retrosternal discomfort for few days

**Systemic complications**

- Fever
- Pneumonitis.

**- Band ligation**

- Endoscopic treatment of choice
- Occludes venous channels



- Effective in less sessions than sclerotherapy
- Same results and less complications than sclerotherapy.

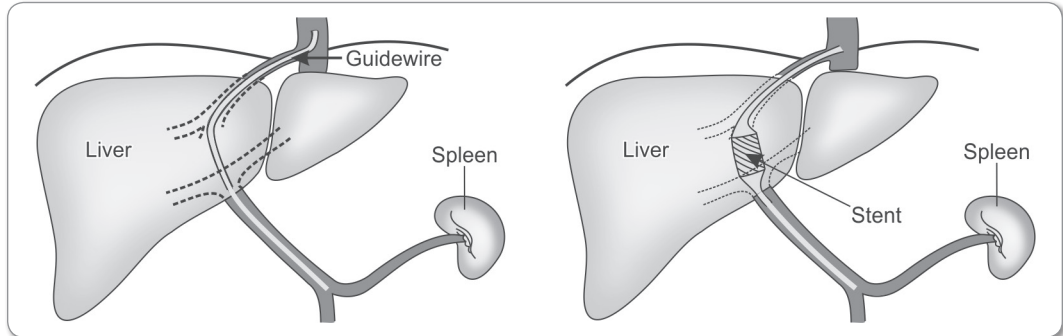
**5. Balloon Tamponade by Sengstaken Blakemore (for esophagogastric varices) or Linton Nachlas Tube (for isolated gastric varices).**

Used when above measures fail to control the bleeding

- The gastric balloon is inflated first by 200 ml of air and pulled upwards to press the gastric fundus
- If bleeding continues, the oesophageal balloon is inflated
- The pressure in the oesophageal balloon should not exceed 40 mm Hg
- This therapy is effective in controlling bleeding in 80–90% of cases
- Can cause esophageal ulceration
- Only used as a temporary measure before/after sclerotherapy or before TIPS/surgery.

**6. TIPS (Transjugular Intrahepatic Portosystemic Stent Shunt)**

- Percutaneously, a connection is created within the liver, between the portal and systemic circulations, to reduce portal pressure in patients with complications related to portal hypertension
- TIPS has replaced operative shunts for managing acute variceal bleeding when pharmacotherapy and endoscopic treatment fail to control bleeding
- Emergency surgical intervention in most centers is reserved for selected patients who are not TIPS candidates.



**Fig. 1: TIPS**

**Indications of TIPS**

- Refractory variceal bleeding
- Refractory ascites
- As a bridge to liver transplant
- 3 or more recurrent episodes of variceal bleeding
- Hepatic hydrothorax

VIATORR is a stent graft used in TIPS whereby, the uncovered part lies in the portal vein and the covered part (3 PTFE layers) traverses the liver parenchyma and the covering prevents bile leak into the graft.

**Complications with TIPS (9 to 50%)**

- Hepatic encephalopathy (15% patients)

- Infection
- Intraperitoneal bleeding
- Subcapsular hematoma
- Hemobilia
- Mortality (30 day) 3 to 13%
- Stent stenosis (more common than thrombosis and is due to neointimal hyperplasia) and thrombosis (33 to 73% patients by the end of 1 year)

### Emergency Surgery

#### Indications

- Refractory variceal bleeding
- Inability to control bleeding by endoscopy
- Hemodynamically unstable patient with bleeding esophageal varices
- Two attempts at endoscopic control followed by rebleeding
- Complications of endoscopy (perforation)
- Hemorrhage from gastric varices or portal hypertensive gastropathy
- Failure of TIPS placement

#### Options

- Esophageal transection with a stapling device
- Splenectomy, portoazygos disconnection and stapling of the oesophagus (**Suigura procedure**)
- Portacaval shunt
- Distal splenorenal shunt in stable patients

**Q. Describe the methods to prevent recurrent variceal hemorrhage in a patient of esophageal varices.**

**Explain the role of surgery in patients with portal hypertension.**

**Ans. Prevention of recurrent variceal hemorrhage**

- Management options include all of the above measures except the tubes and in addition includes two other options—shunt surgeries and liver transplantation
- The treatment is again started same way.
- **Endoscopic therapy with pharmacotherapy is the preferred initial prevention strategy.**

However, because of the rebleeding risk with chronic therapy, subsequent treatment with TIPS, a shunt procedure, a nonshunt operation or liver transplantation should be done.

- Primary indications of TIPS in this setting include
  - Liver transplantation candidates who fail endoscopic and/or pharmacotherapy and are awaiting transplant
  - Advanced hepatic functional decompensation who are unlikely to survive long enough for the TIPS to malfunction
- **Best treatment for patients who have never bled before – propranolol**
- **Prevention of rebleed – sSurgical shunt> TIPS> sclerotherapy> propranolol**

**Surgical therapy of portal hypertension** (include the emergency surgery role from previous question to complete this answer)

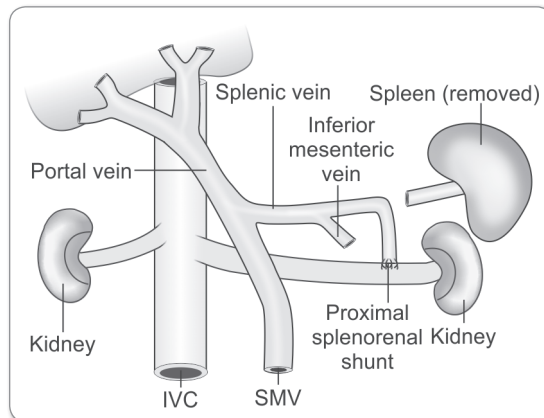
### Shunt surgeries

(**Remember:** For splenic vein, proximal is near SMV-PV confluence and distal is near the spleen. Therefore, spleen is removed in PSRS and never in DSRS. Infact, DSRS is contraindicated in patients with prior splenectomy)

Nonselective (Completely divert portal flow)	Selective	Partial
End to side portocaval shunt (Eck fistula)	Distal splenorenal shunt (Warren)	When the nonselective shunt diameter < 10 mm
Side to side portocaval shunt	Coronary – caval shunt (Inokuchi)	
Interposition graft [portocaval, mesocaval (Drapanas shunt), mesorenal]	Mesenteric left portal bypass (Rex shunt)	
Proximal splenorenal shunt (linton shunt)		

### Nonselective Shunts

- These procedures decompress the splanchnic venous circulation and intrahepatic sinusoidal network
- Side to side nonselective shunts are the most effective shunt procedures for relieving ascites as well as preventing recurrent variceal bleeding
- Both side-to-side and end to side shunts accelerate hepatic failure and lead to frequent postshunt encephalopathy
- Proximal splenorenal shunt consists of anastomosis of the proximal splenic vein (**proximal splenic vein means near the superior mesenteric vein**) to the renal vein. Splenectomy is also performed
- PSRS is slightly less encephalopathic than the other nonselective shunts.



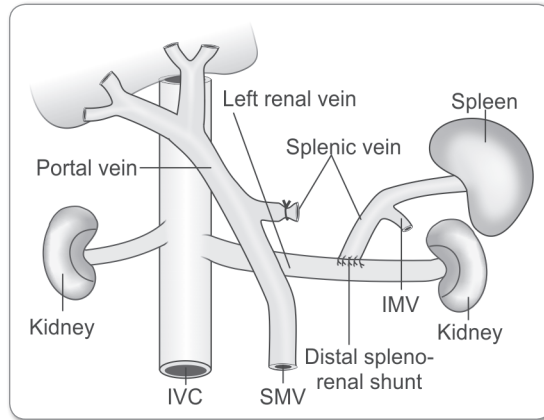


Fig. 2: DSRS and PSRS

### Selective Shunts

#### *The Distal spleno-renal shunt*

- Anastomosis of the distal end of the splenic vein (**near the spleen**) to the left renal vein with interruption of all collateral vessels (e.g. coronary vein and gastroepiploic veins), which connect the superior mesenteric vein and gastrosplenic components of the splanchnic venous circulation
- Results in separation of the portal venous circulation into a decompressed gastrosplenic venous circuit and high-pressure superior mesenteric venous system that continues to perfuse the liver
- A splenic vein diameter less than 7 mm is a relative contraindication to the procedure because of the high incidence of shunt thrombosis in these patients
- No survival benefit over nonselective shunts but a better quality of life in DSRS
- Lower incidence of encephalopathy.

### Hepatic Transplantation

- The only treatment for end-stage hepatic failure
- The only treatment of underlying disease in cases with variceal bleeding and portal hypertension
- Hepatic transplantation should be considered when other complications of cirrhosis develop or hepatic functional decompensation is evident clinically or by careful assessment with quantitative LFTs.

**The most common cause of death in medically treated esophageal varices patients is rebleeding.**

**The most common cause of death in shunted esophageal varices patients is accelerated hepatic failure.**

**Q3. Write a note on achalasia cardia.**

**Describe the management of achalasia cardia.**

**Enumerate the differential diagnoses of achalasia cardia.**

**Differentiate between achalasia and pseudoachalasia.**

**Ans. Introduction**

- It is taken from a Greek word. It means 'failure to relax'. It is basically the failure of lower esophageal sphincter to relax during the deglutition phases.

**Causes**

- Syndromic association with allgrove's disease (alacrima, achalasia, ACTH resistant adrenal insufficiency)
- Destruction of the nerves in Auerbach's plexus to the LES is the primary abnormality.
- Neuromuscular degeneration in the body of the esophagus also occurs.
- Thus, finally both the nerves and muscles are affected.

**Pathophysiology**

- Selective loss of neurons in the Auerbach's plexus that secrete NO and VIP, the inhibitory gut hormones lead to increased tone of LES and failure to relax LES.
- Also causes aperistalsis of the body of esophagus and resultant dilatation of esophagus which when advanced present as sigmoid esophagus.

**Clinical features**

- Men and women are equally affected
- Triad – dysphagia + weight loss + regurgitation
- Dysphagia is more to liquids and progresses slowly.
- Regurgitation of undigested, foul smelling food can lead to aspiration pneumonitis, lung abscess.

**Investigations**

- **Manometry** is the gold standard and shows elevated LES pressure and incomplete LES relaxation with absence of progressive peristalsis in the body of the esophagus and elevated intraesophageal pressure. Simultaneous low amplitude nonperistaltic contractions may be seen in the body.
- **Barium swallow findings**
  - Bird beak/ pencil tip/rat tail appearance
  - Sigmoid esophagus/megaesophagus
  - Absence of gastric air bubble in upright posture.

**Differential diagnoses**

- **Vigorous achalasia** - Achalasia with relatively high esophageal contraction amplitudes (This means that there is activity in the body of esophagus but, this is largely ineffective spasm rather than the true co-ordinated peristalsis), often with minimal esophageal dilation and prominent tertiary contractions on radiographs and with the presence of chest pain.
- **Pseudoachalasia** - Distal esophageal obstruction by tumor, stricture, or surgical manipulation will result in the same functional characteristics as achalasia i.e. narrowing of the distal esophagus with aperistalsis and dilation proximal to the narrowing and has been labeled as secondary achalasia or pseudoachalasia.

**Tumors causing pseudoachalasia include:** Adenocarcinoma of gastric fundus (m.c.), oat cell carcinoma of lung, squamous cell cancer esophagus, lymphoma, prostatic carcinoma metastasis and pancreatic adenocarcinoma.

Achalasia	Pseudoachalasia
Uniform age distribution between 10–70 years	More common after 3rd decade with increased incidence as age increases
In double contrast barium swallow, no morphologic abnormalities are present in classic achalasia	Morphologic abnormalities (stricture) will be present
Amyl nitrate inhalation will cause relaxation of the narrowed part	Amyl nitrate inhalation will not cause relaxation of the narrowed part
Flexible endoscope can be traversed through LES easily without resistance	Resistance is encountered when flexible endoscope is traversed through the lesion. Also, biopsy can be taken to clinch the diagnoses
Pneumatic dilatation is effective	Pneumatic dilatation is ineffective and dangerous and also delays the actual treatment due to false diagnoses.

### Treatment

- Medical management—tried with sublingual nitroglycerine/ nitrates/ calcium channel blockers
- Botulinum toxin injection into LES has also been tried but has a high recurrence rate of 50% within a 6 month post treatment period.
- Procedure of choice—laparoscopic Heller’s myotomy with partial Toupet or Dor fundoplication.
- Indications of esophagectomy—sigmoid esophagus, failure of 2 or more myotomy, undilatable stricture esophagus.
- Newer modality is **peroral endoscopic myotomy (POEM)** which is done under GA using an endoscope with a small 1 cm mucosal incision.
  - It was first done by Parischa
  - **Indications for POEM** as of now—post Heller myotomy failure and sigmoid esophagus
  - After the mucosal incision, a submucosal tunnel is created using methylene blue/ indigo carmine for 10 cm on esophageal side and 5 cm on gastric side
  - The myotomy starts 3 cm below the mucosal incision
  - Once the procedure is done, the mucosal incision is closed.

### Results and prognosis

- Myotomy has a 70% success rate in reliving the symptoms
- Long standing achalasia is a risk factor for squamous carcinoma esophagus.

## Q4. Describe clinical features, types and management of tracheoesophageal fistula.

### Write a note on esophageal atresia.

(**Important: Always Remember:** Tracheoesophageal fistula is congenital as well as acquired. If SN does not mention either specifically, write both... gives credit marks!!!)

### Ans. Esophageal atresia and tracheoesophageal fistula

A tracheoesophageal fistula (TEF) is an epithelialized tract between the esophagus and the trachea.

**Embryological basis**

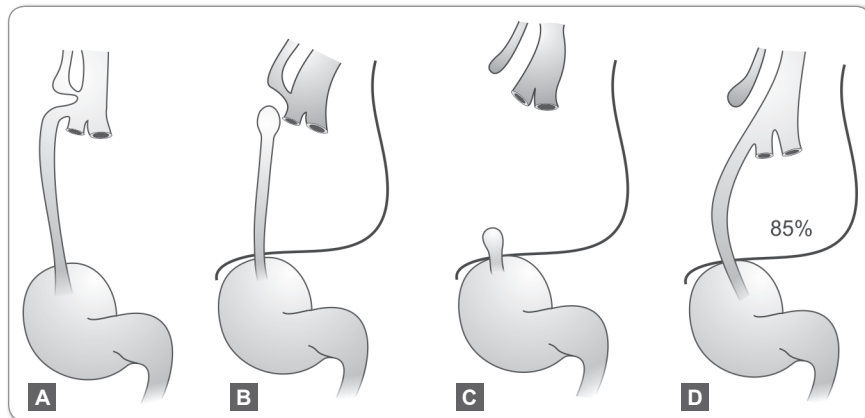
- The division of foregut is the result of fusion of invaginating lateral longitudinal ridges that created a septum dividing the foregut into a dorsal digestive tract and ventral respiratory system
- The formation of tracheoesophageal septum is believed to begin caudally and end cranially
- Most likely origin of TOF is heterogeneous and multifactorial and involves multiple genes and complex gene-environment interactions.

**Theories of EA and TEF**

- Esophageal occlusion and failure of recanalization
- Spontaneous deviation of tracheoesophageal septum
- Abnormal migration of tracheoesophageal septum
- Teratogen: doxorubicin

**“VOGT classification”**

- **Type 1:** Absent esophagus.
- **Type 2:** EA without TEF (TYPE A – 8%)
- **Type 3:** EA with fistula:
  - **a:** EA with proximal TEF (TYPE B – 1-2%)
  - **b:** EA with distal TEF (m.c.) (TYPE C – 84%)
  - **c:** EA with proximal and distal TEF (TYPE D – 4%)
- **Type 4:** Isolated TEF with intact esophagus (TYPE E – 3%)

**Fig. 3:** Tracheoesophageal fistula**Associations (incidence – 50–70%)**

m.c. - Cardiovascular 35%

- **Vacterl association:** 20%
  - Vertebral: 17%
  - Anal 12%
  - Cardiac 20% (m.c. VSD)
  - Renal 16%
  - Limb 5%

- **Syndrome association**

- DiGeorge sequence
- Polysplenia sequence
- Holt-Oram syndrome
- Peirre Robin sequence
- Feingold syndrome
- Fanconi syndrome.

- **Prenatal detection**

- USG - Anechoic area in the middle of the neck, small or absent stomach bubble and associated maternal polyhydroamnios, predictive value 20 to 40%
- Fetal MRI may be a useful adjunct for diagnosis.

- **Postnatal**

- Earliest sign is excessive salivation, typically first feeding is followed by regurgitation, choking and coughing
- Cyanosis with or without feeding, respiratory distress, inability to swallow and inability to pass a feeding tube in the stomach
- If distal fistula present the abdomen distends.

- **Investigations**

- **Plain chest X-ray with a nasogastric tube:** Coiled at upper pouch.
- A few ml of air can be injected through the tube and used as contrast to distend upper pouch as frontal and lateral films taken.
- If necessary 0.5 to 1 ml of diluted barium can be used, if barium used fluoroscopy is advisable to detect proximal fistula. Air in the stomach and bowel confirm distal TEF, absence of air typically represent isolated TEF.
- **Additional testing:**
  - Echocardiography
  - Renal ultrasound
  - Chromosomal analysis

- **Preoperative treatment**

- NPO and IVF 10% D and hypotonic saline
- Start broad spectrum antibiotic and chest physiotherapy
- A sump catheter/double lumen catheter for continuous low pressure negative suction
- Positioning of infants to prevent reflux: Upright sitting/head-up prone position
- Vitamin K analogue should be given.

**Routine ET intubation with elective positive pressure ventilation and elective gastrostomy are to be avoided.**

- **Surgery**

- *Type of Surgery*

- Thoracotomy: Extra-pleural/trans-pleural
- VATS (Video assisted thoracoscopic surgery)



*Approach*

Right sided in case of left aortic arch and left sided in case of right aortic arch

*Techniques*

- Primary closure (single layer increases leaks and double layer increase stricture rates.), if adequate length of esophagus is available
- In case length is not adequate to allow a tension free anastomosis
  - Circular/spiral esophagomyotomy
  - Mobilization of upper pouch and anastomosis
  - Traction sutures and delayed anastomosis after tacking the distal pouch to prevertebral fascia to allow lengthening
  - Cervical esophagostomy with gastrostomy and gastric pull up or colon interposition at an age of 1 year.

*Complication*

- **Early:**
  - Anastomotic leak
  - Anastomotic stricture
  - Recurrent TEF
- **Late:**
  - Gastroesophageal reflux
  - Tracheomalacia
  - Disordered esophageal peristalsis

**A word about acquired tracheoesophageal fistula:***Causes*

- Intubation and tracheostomy tube cuff-related tracheal injury
- Malignant tumors of esophagus and trachea
- Blunt and penetrating trauma, radiation, surgery and caustic ingestion
- Infections, steroid use, hypotension and diabetes are all associated risk factors.

Persistent coughing with meals, frequent respiratory infections including pneumonia are common with larger fistulas.

**Investigations** include endoscopy, bronchoscopy, and barium esophagogram.

*Management*

- **Initial:** IV antibiotics, gastrostomy for feeding and esophageal stent to decrease contamination of lungs.
- **Surgery: Three main steps**
  - **Step 1:** Exposure of fistula tract
  - **Step 2:** Excision of the involved trachea and primary repair of esophagus
  - **Step 3:** Muscle flap interposition between trachea and esophagus to prevent recurrence and primary trachea repair

**Q5. Write a note on Barrett's esophagus.**

**Discuss management of gastroesophageal reflux disease.**

**Ans. Gastroesophageal reflux disease and Barrett's esophagus**

It is a disease characterized by classic triad of epigastric pain, retrosternal pain and regurgitation.

*Pathophysiology*

- **Lower esophageal sphincter (LES)**

Mainly formed by

- Intrinsic musculature of esophagus
- Sling fibers of cardia
- Crura of diaphragm
- Intra-abdominal length of esophagus

- **Normal values at LES**

- Total length—> 2 to 5 cm
- Intra-abdominal length—> 1 cm
- Resting pressure—> 6 mm Hg to 26 mm Hg
- Acid reflux episodes—4–7%

- **Defective LES**

- Total length < 2 cm
- Intra-abdominal length < 1 cm
- Resting pressure < 6 mm Hg
- Acid reflux episodes—12% in esophagitis and upto 26% in Barrett's esophagus

*Chain of progression*

GERD – Intestinal type metaplasia (Barrett's esophagus) low grade dysplasia, high grade dysplasia, 40 times increased risk of adenocarcinoma in this 10% population (incidence of Barrett's)

*Clinical features*

- **Classic triad** of symptoms or any combination of it can be seen.
- Occasional patient may present with hoarseness and change in voice and in severe cases, complete loss of voice due to severe reflux induced laryngeal edema and laryngitis. These symptoms when mild characteristically show **diurnal variation** with symptoms more in morning and decreased by evening.

*Diagnoses*

- UGI endoscopy and biopsy of suspicious areas
- Endoscopic ultrasound and biopsy, chromoendoscopy (indigo carmine, methylene blue, toluidine blue), laser induced endoscopy (5-aminolevulinic acid), optical coherence tomography can also be used
- Manometry
- 24 hour pH monitoring (Gold Standard)
  - Document number of reflux episodes (pH < 4)
  - Longest reflux episode
  - Number of reflux episodes > 5 minutes
  - Extent of reflux
- Demeester score (N < 14.7)

*Management***GERD***General measures*

- Lifestyle modifications
- Cessation of smoking and alcohol
- Avoid large, fatty meals at sleep time atleast before 2 hrs
- Medical management include double dose proton pump inhibitors.

*Indications of surgery*

- Failure of medical therapy
- Complicated GERD (Barrett's, peptic stricture, esophagitis, ulcer)
- Persistent symptoms at a young age.

*Indications of primary surgery in GERD (contraindications to medical management)*

- Supine reflux/bile reflux
- Structurally defective sphincter
- Erosive esophagitis
- Poor esophageal contractility.

*Procedures*

- Allison procedure—anatomical repair
- Angelchick prosthesis—horseshoe shaped prosthesis at GE junction
- Transthoracic procedures—Nissen, belsey, collis gastroplasty

*Types of fundoplication*

- Watson (90° anterior)
- Toupet (180° anterior)
- Dor (180° posterior, now 270° posterior)
- Belsey mark 4 (270° anterior)
- Nissen (360°)
- Rosette and hill modification of nissen (360° using only the anterior stomach)
- Laparoscopic Nissen (**Gold Standard**)

**Long term failure rate**—25%

*Indications for a partial fundoplication in GERD*

When peristalsis is absent or severely disordered or when amplitude < 20 mmhg, 2/3 fundoplication should be done.

*Indications of esophageal resection*

- Global absence of contractility with short esophagus
- Several failed antireflux procedures
- >50% interrupted or dropped contractions with short esophagus

*Management of Barrett's esophagus*

- Columnar epithelium lining distal esophagus
- Biopsy showing columnar metaplasia

<b>Intestinal metaplasia</b>	<b>Endoscopy and biopsy every 2 years</b> (This is a 4 quadrant biopsy at 2 cm intervals starting 1 cm from GE junction)
<b>Low grade dysplasia</b>	<b>Endoscopy and biopsy every 6 months</b> (This is a 4 quadrant biopsy at 2 cm intervals starting 1 cm from GE junction)
<b>High grade dysplasia</b>	<b>Endoscopic mucosal resection &gt; esophagectomy</b> Other options – photodynamic therapy, thermal ablation, electrocoagulation

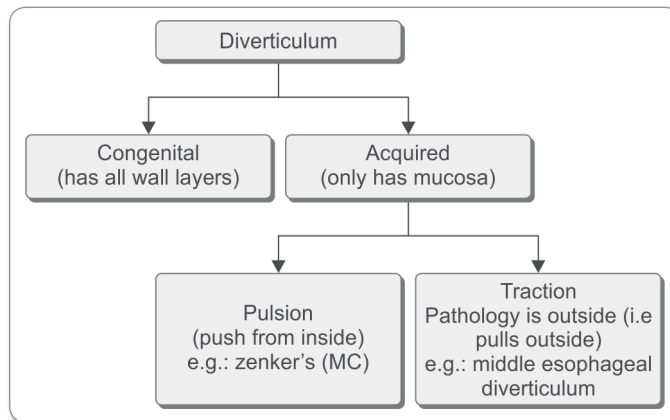
**Q6. Describe the clinical features and management of a patient with zenker's diverticulum.**

**Classify esophageal diverticula and describe zenker's diverticulum.**

**Ans. Esophageal diverticula**

The three most common sites of occurrence are:

- Pharyngoesophageal (zenker's)
- Parabronchial (midesophageal)
- Epiphrenic (supradiaphragmatic)



#### Classifications

1.	Congenital and acquired	
2.	True False	Contain all layers Only mucosa and submucosa
3.	Pulsion  Traction	Occur because of elevated intraluminal pressure and are false For example – Zenker/Epiphrenic External inflammatory mediastinal lymph nodes adhering to esophagus

#### Pharyngoesophageal (Zenker's) diverticulum

- Also called **cricopharyngeal achalasia**
- Most common of all esophageal diverticula
- Found herniating into Killian's triangle, between the oblique fibers of the thyropharyngeus muscle and the horizontal fibers of the cricopharyngeus muscle

- As the diverticulum enlarges, the mucosal and submucosal layers dissect down the left side of the esophagus into the superior mediastinum posteriorly along the prevertebral space.

#### *Clinical features*

- Older patients, seventh decade of life
- Initially asymptomatic
- Chronic cough, excessive salivation and intermittent dysphagia
- Regurgitation of foul-smelling, undigested material
- Voice changes, retrosternal pain and respiratory infections
- The most serious complication from an untreated Zenker's diverticulum is aspiration pneumonia or lung abscess.

#### *Diagnosis*

- Barium esophagography (lateral views)
- At the level of the cricothyroid cartilage, the diverticulum can be seen filled with barium resting posteriorly alongside the esophagus.

#### *Treatment*

- Endoscopic or open surgical Dohlman's procedure.
- Small (<2 cm diverticulum) – myotomy (thyropharyngeus and cricopharyngeus muscles) alone
- In frail patients—Diverticulopexy without resection with myotomy
- Large sac (>5 cm)—excision of the sac (Diverticulectomy) with myotomy is indicated or Endoscopic division of the common wall between the esophagus and diverticulum using a laser or stapler has also been successful. The endoscopic technique has gained favor and is advocated for patients with diverticula between 2 and 5 cm
- For diverticula 3 cm or less in size, surgical repair is superior to endoscopic repair in eliminating symptoms
- For any diverticulum larger than 3 cm, the results are the same.

#### **Midesophageal traction diverticula**

- Inflamed mediastinal lymph nodes from an infection with tuberculosis, histoplasmosis and resultant fibrosing mediastinitis exerts traction on the wall of the esophagus and leads to the formation of a true diverticulum in the midesophagus
- Some may also be caused by a primary motility disorder, such as achalasia, diffuse esophageal spasm (DES), or nonspecific esophageal motility (NEM) disorder
- Symptomatic / >2 cm size – diverticulopexy with a long esophagomyotomy if patient has a concomitant motility disorder.

#### **Epiphrenic Diverticula**

- Found adjacent to the diaphragm in the distal third of the esophagus, within 10 cm of the GEJ
- Pulsions, or false, diverticula that are often associated with DES, achalasia
- More common on the right side and tend to be wide-mouthed
- Similar to that of a midesophageal diverticulum
- If a large hiatal hernia is also present, the diverticulum is excised, a myotomy performed, and the hiatal hernia repaired. Failure to repair the hernia results in a high incidence of postoperative reflux.

Other diverticula – Killian Jamieson diverticula—occurs inferior to cricopharyngeus.

**Q7. Discuss the management of esophageal perforation.****Enumerate the causes of esophageal perforation and discuss its treatment.****Ans. Esophageal perforation***Causes*

- Most common cause – Iatrogenic perforation (endoscopic instrumentation for a diagnostic or therapeutic procedure, endoscopic dilation, stent placement, difficult endotracheal intubation, blind insertion of a minitracheostomy and injury during dissections in the neck, chest, and abdomen)
- Boerhaave's syndrome (Postemetic rupture of the esophagus. Other predisposing factors include blunt thoracic trauma, epileptic seizures, defecation and childbirth)
- Foreign body ingestion
- Trauma

Most common site of perforation is left lateral posterior into mediastinum or just above GE junction

*Symptoms and signs*

- Neck, substernal or epigastric pain
- Vomiting, hematemesis
- Cervical perforations may present with neck ache and stiffness
- Thoracic perforations present with shortness of breath and retrosternal chest pain lateralizing to the side of perforation
- Abdominal perforations present with epigastric pain that radiates to the back if the perforation is posterior
- Hemodynamic instability and shock
- Subcutaneous air in the neck or chest
- Shallow decreased breath sounds

*Diagnoses*

- **Elevated white blood cell count and an elevated salivary amylase level** in the blood or pleural fluid
- **Chest roentgenogram** may demonstrate a hydropneumothorax
- **Contrast esophagography** with a film in the left lateral decubitus position is performed using barium for a suspected thoracic perforation and Gastrografin for an abdominal perforation.

(IMP : water soluble nonionic contrast media (iopamidol) is used in thoracic perforation which is inert and nontoxic to thorax. If no leak here, barium is done to confirm. In abdominal perforations, barium is contraindicated as it causes barium peritonitis. Here, ionic water soluble contrast media like gastrograffin is used whenever perforation is suspected in abdomen. This is not used in thorax as in thorax it can cause chemical pneumonitis and mediastinitis. Whereas barium even if aspirated causes only inert granulomas and is therefore safe to use in thorax. The same ionic contrast media is used in cases of meconium syndromes)

- Chest CT scan—mediastinal air and fluid at the site of perforation
- Surgical endoscopy—if the esophagram is negative or if operative intervention is planned. Mucosal injury is suggested if blood, mucosal hematoma or a flap is seen or if the esophagus is difficult to insufflate.

*Treatment*

- Resuscitation, urinary catheter and secured airway
- IV fluids and broad-spectrum antibiotics
- NPO and nutritional access needs.

*Indications for nonoperative management*

Cameron criteria include

- Mild symptoms (stable patient)
- No evidence of sepsis
- When the leak is draining back into esophagus in barium study (contained perforation).

*Indications for surgery*

- Persistence or progression of the perforation
- If a patient's clinical condition deteriorates or
- Perforation is no longer contained.

*Approach*

- Cervical perforations—neck incision on the same side of the perforation
- Thoracic perforations—right chest for the upper two thirds of the esophagus in 4th intercostals space for perforation above carina and 6th intercostals space for the perforation below carina and the left 7th intercostal space for the lower third
- Abdominal perforations are approached from the left chest or abdomen. Repair is buttressed with a pleural patch or fundoplication.

**In an unstable patient with a contained perforation** a temporary stent may be placed and conservative measures initiated.

**In an unstable patient with a free perforation**, surgical intervention with débridement of devitalized tissue, esophageal diversion or resection, creation of an esophagostomy, wide drainage, placement of a gastrostomy, and feeding jejunostomy is indicated.

*The importance of time (the golden period)*

- When patients present within 24 hours of perforation, inflammation is generally minimal and primary surgical repair is recommended. However, it is by no means a definite cutoff time. Primary repair of the perforation is acceptable at any time
- All repairs are buttressed with healthy tissue flaps and widely drained.

**If primary repair or the muscle flap fails** - resection or exclusion of the esophagus with a cervical esophagostomy, gastrostomy, feeding jejunostomy and delayed reconstruction is recommended.

**Resection** is recommended for patients with mid-to high-level perforations. Resection with diversion is recommended in resectable carcinoma, megaesophagus from end-stage achalasia, severe peptic strictures or a history of caustic ingestion. Primary repair is contraindicated in these situations if the cause cannot be corrected entirely.

**Exclusion** is recommended for low perforations in which esophageal salvage is possible or in any unstable patient for whom resection would not be tolerated.

**Q8. Discuss in brief the features and management of caustic/ corrosive injury/ ingestion in upper GIT.****Ans. Corrosive injury to upper GIT***Incidence*

- Most common mode in children—accidental
- Teenagers and adults—suicidal, large amounts, more serious

*Agents*

- Acid—HCl,  $H_2SO_4$ , oxalic and phosphoric acid
- Alkali – NaOH, KOH,  $Na_2CO_3$
- Ammonia, detergents

*Pathogenesis*

- Acid—coagulation necrosis  
Cause pylorospasm and therefore, more injury to stomach especially in prepyloric region
- Alkali—Liquefaction necrosis  
More injury to esophagus, however, it is more dangerous if in stomach.

*Phases of alkali injury*

- Phase 1—Necrosis (1-4 days)
- Phase 2—Granulation (5-15 days) – esophagus is at its weakest stage
- Phase 3—Cicatrization (3 week onwards).

*Clinical presentation*

- Oropharyngeal pain (more with acid)
- Respiratory symptoms—hoarseness, dyspnea
- Retrosternal pain, hematemesis, vomiting
- Chest and back pain, diffuse abdominal pain in case of intraabdominal perforation
- Late manifestations – GERD, stricture, GOO, malignancy related symptoms.

*Diagnoses*

- Endoscopy within 12 to 24 hours
- Contraindications to endoscopy
  - Absolute—suspected esophageal perforation/airway obstruction
  - Relative—5 to 15 days after surgery/third degree burn of hypopharynx or glottis edema
- Endoscope should not go beyond the proximal limit of present injury when done within 12 to 24 hours.
- Distal extent can be evaluated at a later stage or by CT.

*Endoscopic grading*

- Mucosal edema and erythema
- Mucosa and submucosa involvement
  - Superficial erosion, exudates, ulcer
  - Deep erosion, exudates, ulcer
- Transmural
  - Focal necrosis
  - Extensive necrosis
- Perforation



CT scan is the investigation of choice when needed in 5 to 15 days duration

CT grading

- Mucosal lesion, rest wall normal
- Esophageal wall swelling, periesophageal soft tissue normal
- Wall edema, soft tissue infiltrate, well preserved interface
- Lost interface with fluid collection around the esophagus or descending aorta.

*Management*

- **Acute phase**
  - Resuscitation
  - Fiberoptic intubation
  - IV fluids as in burns
  - IV antibiotics
  - Aerosolised steroids to be used only if there is an airway involvement
  - Emergency surgery indications
    - Esophageal perforation, mediastinitis
    - Gastric perforation
    - Interstitial air in wall of stomach
    - Alkaline aspirates from stomach
  - Emergency procedure—cervical esophagostomy with gastrostomy and feeding jejunostomy. All necrosed organs should be resected at surgery. However, esophageal resection is a negative survival predictor.
  - 1st and 2nd degree—observation and orally allowed when saliva can be swallowed painlessly
  - Neutralization, lavage, activated charcoal, steroid for stricture prevention have no role in management
- **Chronic phase**

*Management of complications*

- **Stricture** – Dilatation daily for 3 weeks f/b alternate day for 3 weeks f/b weekly for 3 months can be done by pneumatic/bougie or stent dilatation in antegrade or retrograde manner.

Rate of stricture formation is upto 60% by 1 month and it gets completed in 100% patients by 8 months. Therefore, definitive surgery is planned at 8–12 months.

Esophageal resection with gastric or colonic conduit in posterior mediastinum is the procedure of choice.

- **GOO** (gastric outlet obstruction)
  - Endoscopic gastric dilatation is not an option.
  - Gastrojejunostomy with surveillance for Gastric cancer
  - Partial or total gastric resection
  - Pyloroplasty has more recurrence rates and therefore not preferred.
- **Tracheoesophageal fistula** repair
- **Malignant transformation** to be managed as non corrosive malignancy. However, prognosis of these is better than sporadic ones.

## STOMACH AND DUODENUM

**Q9. Describe the lymphatic drainage of the stomach and give its clinical importance.**

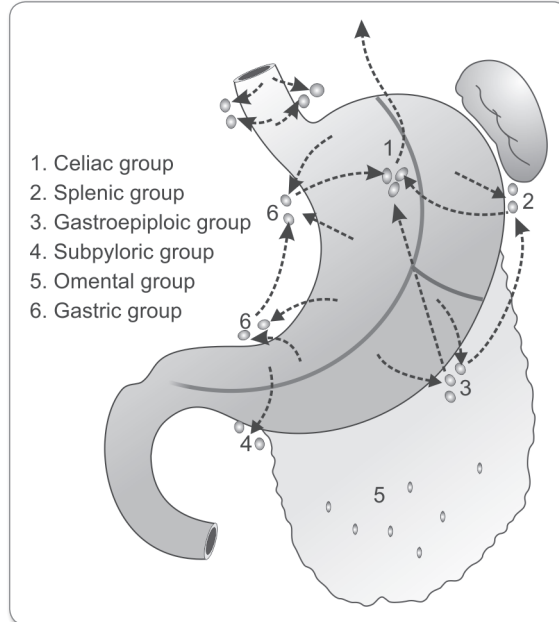
**Describe the blood supply and lymphatic drainage of stomach.**

**Ans.**

### Lymphatic drainage of stomach

Coller et al. outlined four drainage zones of the gastric lymphatics. Most of the lymphatic drainage of the stomach finds its way to the celiac nodes.

- **Zone I (inferior gastric nodes)**—are the nodes around the **right gastroepiploic artery** and **gastrooduodenal arteries** to the nodes around hepatic artery and celiac nodes. These drains into the subpyloric and omental nodes.
- **Zone II (splenic)** are the nodes around **left gastroepiploic and short gastric arteries** to pancreaticosplenic nodes to splenic artery nodes to celiac nodes and also drains into the pancreaticosplenic nodes.
- **Zone III (superior gastric)** are nodes around the **left gastric artery** and drains into the superior gastric nodes and finally into celiac nodes.
- **Zone IV (hepatic)** are the nodes around the **right gastric artery** and drains into the suprapyloric nodes and finally to the celiac nodes.



**Fig. 4:** Lymphatic drainage of stomach

### Clinical applications

- Both anatomic location and number of node metastases are important predictors of survival in gastric cancer patients.

- In early gastric cancer (protruded type) of the lower one-third of the stomach, metastasis is to the lymph nodes near the lesion. If cancer with muscularis propria involvement occurs, distant lymph nodes were found to be involved with the cancer
- Among the cases with lymph node metastasis, differentiated early gastric cancer had more lymph node involvement and wider extent of metastasis than undifferentiated cancers. The embryology of the stomach and related organs is such that the body and tail of the pancreas (derived from the dorsal pancreatic anlage), together with the spleen lie in the dorsal mesogastrium. They share both a common blood supply (left gastric and splenic arteries) and a common lymphatic drainage with the proximal portion of the stomach.
- The head of the pancreas (derived from the ventral pancreatic anlage) lies in the mesoduodenum. The pancreatic head shares its blood supply (pancreaticoduodenal and gastroduodenal arteries) and lymphatic drainage with the duodenum, the distal common bile duct and the distal stomach.
- Theoretically, cancer of the proximal stomach can be effectively treated by en bloc resection of the organs supplied by the left gastric and splenic arteries:
  - distal esophagus
  - proximal two-thirds of the stomach and greater omentum
  - spleen
  - body and tail of the pancreas
- Cancer of the distal stomach can be treated by en bloc resection of the organs supplied by the common hepatic artery, sparing, of course, the artery itself:
  - head of the pancreas
  - distal stomach and greater omentum
  - duodenum
  - distal bile duct

Cancer of the stomach may metastasize not only to the supraclavicular or scalene nodes but also to other areas such as the axillary region.

#### **Lymph node stations in stomach cancer**

1. Right cardiac
2. Left cardiac
3. Lesser curvature
4. Greater curvature
5. Suprapyloric
6. Infrapyloric
7. Left gastric
8. Common hepatic
9. Celiac
10. Splenic hilum
11. Splenic artery
12. Hepatoduodenal ligament
13. Retropancreatic
14. Mesenteric root

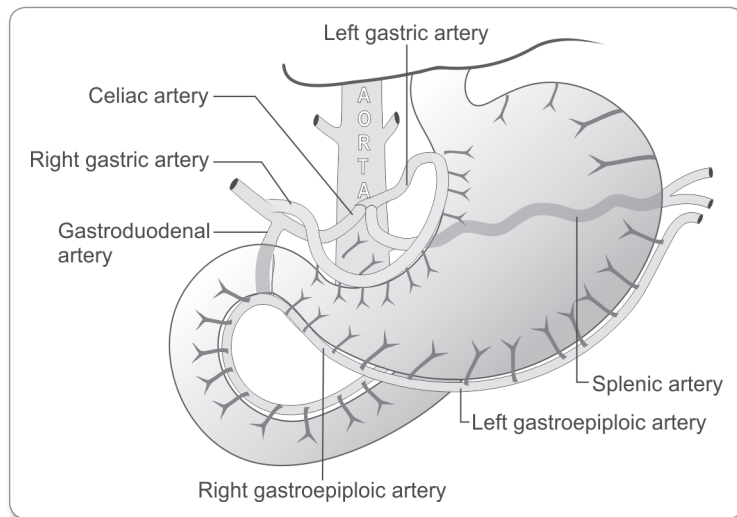
15. Transverse mesocolon

16. Paraaortic

Minimum number of lymph nodes to be resected in carcinoma stomach surgery—15.

**Blood supply to stomach is as follows:**

<b>Celiac Artery</b>	<b>Splenic artery</b>	<ul style="list-style-type: none"> <li>• Left gastroepiploic artery</li> <li>• Short gastric arteries</li> <li>• Posterior gastric artery</li> </ul>
	<b>Common hepatic artery</b>	<ul style="list-style-type: none"> <li>• Right gastric artery</li> <li>• Right gastroepiploic artery from gastroduodenal artery</li> </ul>
	<b>Left gastric artery (most important)</b>	



**Fig. 5:** Blood supply of the stomach

The gastroepiploic arteries supply the greater curvature and adjacent stomach whereas the gastric arteries supply the lesser curvature and adjacent stomach. Three of these four main vessels can be sacrificed without significant consequences. Venous drainage follows the arterial pattern.

**Q10. Write a note on the physiology of gastric acid secretion.**

**Ans.** Gastric acid is produced by a proton pump in the parietal cell.

**Basal acid secretion**

- Occurs in a circadian pattern with highest levels occurring during the night and lowest levels during the morning hours.
- Factors affecting acid secretion :
  - Increase by histamine, acetylcholine, gastrin
  - Decrease by somatostatin, gastric inhibitory peptide, prostaglandin, secretin
- Cholinergic input via the vagus nerve and histaminergic input from local gastric sources are the principal contributors to basal acid secretion

**Stimulated acid secretion**

- Occurs primarily in three phases based on the site where the signal originates.

**1. Cephalic phase**

- It results from sight, smell, thought or taste of food even before food enters stomach
- Neurogenic signals of gastric secretion arise in cerebral cortex and in appetite centers of amygdala and hypothalamus
- The afferent fibers travel through vagus nerve. This parasympathetically(vagally) mediated phase can be conditioned. Tension, anger and hostility increase gastric secretion whereas fear and depression decreases it
- It accounts for 20% of gastric secretion.

**2. Gastric phase**

- Once the food enters the stomach it excites:  
Vagovagal reflex (from stomach to brain)  
local enteric reflex  
gastrin mechanism
- When nutrients (amino acids and amines) enters the stomach they directly stimulate the G cell to release gastrin, which in turn activates the parietal cell via direct and indirect mechanisms
- Distention of the stomach wall also leads to gastrin release and acid production
- It accounts for 70% of gastric secretion.

**3. Intestinal phase**

- In this phase the presence of food in the upper portion of the small intestine particularly in the duodenum cause secretion of small amounts of gastric juice in the stomach because of small amounts of gastrin released by the duodenal mucosa.

**Clinical correlation**

Drugs affecting gastric acid production and secretion:

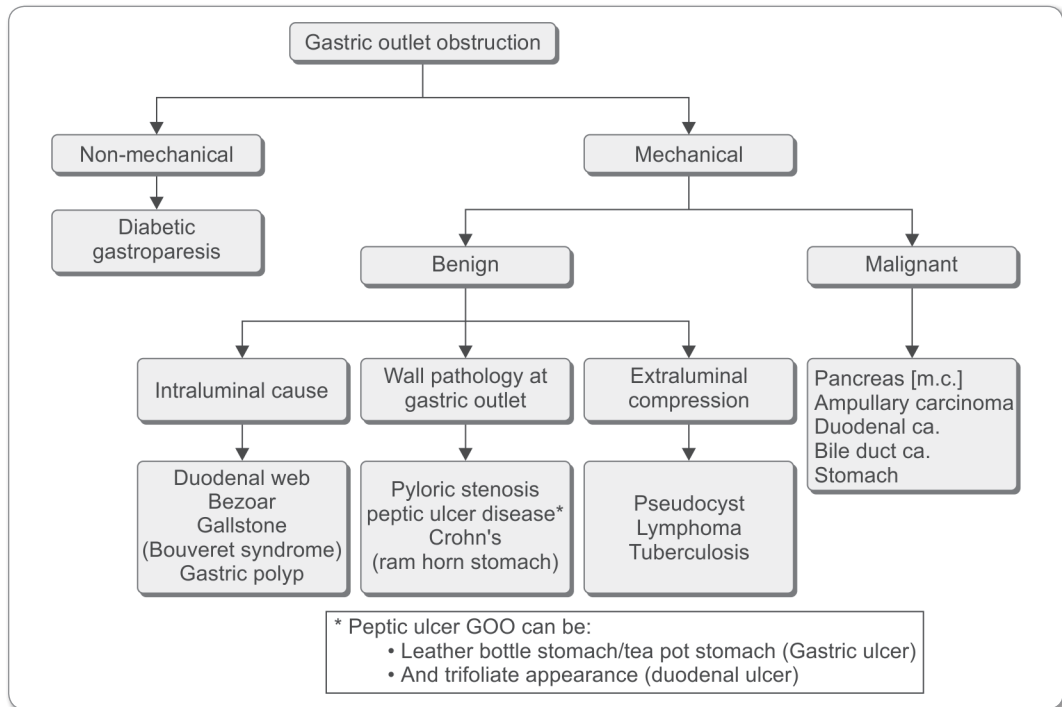
- H<sub>2</sub> blockers: Reversible block of histamine H<sub>2</sub>-receptors, decrease H<sup>+</sup> secretion by parietal cells
- Proton pump inhibitors: Irreversibly inhibit H<sup>+</sup>/K<sup>+</sup> ATPase in stomach parietal cells
- Misoprostol: A PGE<sub>1</sub> analogue. Increase the production and secretion of gastric mucous barrier.

**Q11. Enumerate the causes of gastric outlet obstruction.**

**Describe the pathophysiology of gastric outlet obstruction and discuss the fluid and electrolyte abnormalities in a patient with gastric outlet obstruction.**

- Ans.** Gastric outlet obstruction is a clinicopathological entity resulting from any disease or disorder that leads to a mechanical or non-mechanical impairment in gastric emptying function.

**Causes** are as shown in the flow chart below:



### Clinical Presentation

- Vomiting is the principal symptom with undigested food particles and nonbilious nature of vomitus usually within 1 to 2 hrs of food intake
- Progressive weight loss and malnutrition
- Early satiety, bloating, anorexia, epigastric fullness are other symptoms
- On examination, patient may be dehydrated and malnourished with presence of a tympanitic mass in the epigastrium and left hypochondrium with presence of succussion splash.

### Metabolic complications of gastric outlet obstruction

#### Pathophysiology

- Loss of acid in gastric vomiting lead to hypochloremic metabolic alkalosis
- Initially  $H^+$  is exchanged for  $Na^+$  and  $K^+$  in distal tubules of kidney to conserve  $H^+$ . Therefore, the urine initially is alkaline with resultant hypokalemic, hyponatremic, hypochloremic metabolic alkalosis
- In longstanding cases, due to prolonged and severe hypochloremia, kidney starts conserving chloride ions which are absorbed along with  $Na^+$  ions which lead to loss of potassium and hydrogen ions in urine thus resulting in paradoxical aciduria
- Thus, paradoxical aciduria is a consequence of renal attempt to correct hypochloremia

- So final result is **hypokalemic, hyponatremic, hypochloremic metabolic alkalosis with paradoxical aciduria**.

### Diagnoses

- **Sodium chloride load test:** First nasogastric tube is inserted and contents aspirated. If contents greater in amount then no need to perform the test. Otherwise, to confirm GOO, 750 ml of isotonic saline is instilled into stomach via nasogastric tube and effluent is checked. If volume > 400ml is aspirated at 30 minutes after instillation, the test is positive
- **Nuclear gastric emptying studies, barium upper GI studies, Upper GI endoscopy** can also be used as indicated
- **CECT abdomen** can also aid in diagnoses especially when periampullary or pancreatic pathology is suspected.

### Management

- Nasogastric tube is inserted to decompress the stomach and GI fluid loss is replaced ml for ml
- Percutaneous jejunostomy can be done for feeding purpose if long standing GOO
- This condition is more a medical emergency than a surgical emergency where patient presents with fluid and electrolyte abnormalities
- Correct the abnormality with 0.45% saline and 5% glucose at 150 to 175 ml/kg/day with 2 to 4 meq/kg potassium added to the fluid for the initial 24 hours
- Maintain a urine output of >1 ml/kg/hr
- Serum bicarbonate also needs normalization before proceeding to surgery for correction of cause
- Post operative IV fluids should be continued for several hours followed by oral intake as tolerated.

**The surgical management** depends on correction of the underlying condition that is causing GOO.

### Q12. Write a note on congenital hypertrophic pyloric stenosis.

**Mention the fluid and electrolyte abnormalities in congenital hypertrophic pyloric stenosis – same as in gastric outlet obstruction.**

**Ans.** A condition characterized by:

- Mechanical obstruction of gastric outlet due to hypertrophy of the circular muscle layer of the pylorus of the stomach
- The pyloric canal lengthens, the whole pylorus thickens and the mucosa becomes oedematous causing functional obstruction of the gastric outlet.

### Clinical presentation:

- **Incidence :** 1 in 4000 live births
- First born males are the more commonly affected infants
- High risk in offsprings of parents with this condition.

### Symptoms

- Vomiting is the first symptom in most children
  - May occur after every feeding or only after some feedings.

- Usually starts around 3 weeks of age, but may start any time between 1 week and 5 months of age.
- Forceful (projectile vomiting)
- The infant is hungry after vomiting and wants to feed again.

*Other symptoms generally appear several weeks after birth and may include:*

- Dehydration gets worse with the severity of the vomiting. Prolonged vomiting leads to the loss of large quantities of gastric secretions rich in  $H^+$  and  $Cl^-$ .
- As a result of dehydration, the kidney attempts to conserve  $Na^+$  to maintain volume, by exchanging them for  $K^+$  and  $H^+$  (paradoxical aciduria).
- Starvation can exacerbate diminished hepatic glucoronyl transferase activity, and indirect hyperbilirubinemia may be seen in 1–2% of affected infants.
- Failure to gain weight or weight loss.
- Wave-like motion of the abdomen shortly after feeding and just before vomiting occurs.

### **Etiology**

- It has been found in IHPS specimens that the muscle layer is deficient in:
  - The quantity of nerve terminals
  - Markers for nerve-supporting cells
  - Peptide-containing nerve fibers
- This abnormal innervation of the muscular layer leads to failure of relaxation of the pyloric muscle, increased synthesis of growth factors and subsequent hypertrophy, hyperplasia and obstruction
- There is an increased incidence of IHPS in infants receiving erythromycin. The reason is unclear, although a prokinetic effect on gastric muscle contraction is postulated.

### **Diagnosis**

- Initially suggested by the typical clinical presentation  
In physical examination : The mass is firm, mobile, approximately 2 cm, best palpated from the left, located in the midepigastrium beneath the liver edge
- Palpation of the hard muscle mass or olive is diagnostic in conjunction with a typical history.
- Diagnosis by palpation of olive have only 49% successful of cases in recent years compared with 78% successful cases 30 years ago.
- Ultrasonography is used to measure the thickness of the pyloric wall and the length of the pyloric canal.
  - normal wall thickness < 2 mm, IHPS > 4 mm
  - normal length of the pyloric canal < 10 mm, IHPS > 15 mm and
  - outer wall to wall diameter > 15 mm
- Sensitivity and specificity as high as 100%
- Signs on barium meal – String sign, shoulder sign and double- track sign.

### **Differential Diagnoses**

- Gastroesophageal reflux
- Adrenal insufficiency
- Viral gastroenteritis



**Treatment**

**The preoperative treatment** is directed toward correcting the fluid, acid-base and electrolyte.

Losses as CHPS like GOO is more a medical emergency.

- Intravenous fluid therapy is begun with 0.45 to 0.9% saline, in 5 to 10% dextrose with the addition of potassium chloride in concentrations of 30 to 50 mEq/L
- Fluid therapy should be continued until the infant is rehydrated and the serum bicarbonate concentration is less than 30 mEq/dL, which implies that the alkalosis has been corrected
- Most infants can be rehydrated within 24 hours.

**Surgical management**

- Once resuscitated the infant can undergo the **Fredet-Ramstedt pyloromyotomy**, which is the procedure of choice
- It consist of incision in to the sphincter muscle of pylorus
- NG tube is passed and gastric content are aspirated just prior to surgery.

**Complications**

- Wound infection, mucosal perforation at duodenal end and incomplete myotomy.

**Q13. Discuss the role of *H. pylori* in pathogenesis and management of peptic ulcer disease.**

**Ans.**

**Helicobacter pylori Infection**

- Warren and Marshall were the first to identify and isolate the organism.
- 90% of duodenal ulcers and roughly 75% of gastric ulcers are associated with *H. pylori* infection.

**Organism characteristics**

- It is a spiral or helical gram-negative rod with 4 to 6 flagella.
- It is microaerophilic and the optimal temperature for isolation is 35°C to 37°C with growth occurring after 2 to 5 days.
- It resides in gastric-type epithelium because only this epithelium expresses the necessary adherence receptors for the *H. pylori* to attach and reside within or beneath the mucus layer which protects it from both acid and antibiotics.
- It is one of the most potent producers of urease which splits urea into ammonia and bicarbonate creating an alkaline microenvironment in the setting of an acidic gastric milieu. It also produces catalase and oxidase.
- It can also be found in heterotopic gastric mucosa in proximal esophagus and Barrett's esophagus, in gastric metaplasia in the duodenum and Meckel's diverticulum and in heterotopic gastric mucosa in the rectum.
- It can be **grown in** chocolate medium and skirrow's medium.

**Mode of infection**

- **Infection is more common in developing countries, has familial clustering and occurs through contaminated food and water**

- A person once infected will never show spontaneous remission, and once treated will show eradication of the organism and no ulcer recurrence.

### Mechanism of injury

- **The mechanisms** responsible for *H. pylori*-induced GI injury remain to be fully elucidated, but three potential mechanisms have been proposed:
  - Production of toxic products (ammonia, cytotoxins, mucinase and phospholipase) to cause local tissue injury
  - Induction of a local mucosal immune response
  - Increased gastrin levels with a resultant increase in acid secretion. The toxic products cause mucosal injury and thrombosis in the microcirculation
- *H. pylori* is known to cause a local inflammatory reaction in the gastric mucosa and to produce chemotactic factors that attract neutrophils and monocytes. Activated monocytes and neutrophils in turn, produce a number of proinflammatory cytokines and reactive oxygen metabolites
- Reduction in antral D cells and decreased somatostatin caused by infection with *H. Pylori* also predispose to ulceration.

### Diagnostic methods

#### Noninvasive Techniques

- **Blood test:** Serology. Not useful to document eradication
- **Urea breath test:** The patient swallows a capsule, liquid or pudding that contains urea "labeled" with a special carbon atom. After a few minutes, the patient breathes into a container, exhaling carbon dioxide. If the carbon atom is found in the exhaled breath, *H. pylori* is present, as this bacterium contains large amounts of urease, a chemical that breaks urea down into carbon dioxide and ammonia.
- **Stool antigen test:** The patient provides a stool sample, which is tested for *H. pylori* antigens.

#### Invasive Techniques

- **UGI endoscopy and biopsy based tests**  
Rapid urease test/histology (warthin starry silver stain, giemsa stain)/culture can all be done on the sample.

### Diseases associated with *H. pylori* infection

- Duodenal ulcer
- MALT lymphoma
- Antral gastritis/ Chronic atrophic gastritis/Nonatrophic pangastritis
- Gastric adenocarcinoma
- Gastric ulcer
- Carcinoma pancreas, gallbladder, squamous cell cancer esophagus
- Menetrier's disease.

### Management implications

- All patients are given anti-*H. pylori* medications as the standard triple drug therapy [lansoprazole/omeprazole + metronidazole + clarithromycin (LCM)] for **14 days** with urea breath test as the best follow up test to document eradication

- Also patients with low grade MALT lymphoma are prescribed the anti-*H. pylori* medication and response is evaluated. This might be the only treatment required in these cases
- Recurrence of peptic ulcer without this medical management is in the range of 60 to 70% which is decreased to less than 10 to 20% with the medical management.

**Q14. Classify Peptic ulcers. Give the surgical options for management of peptic ulcers.**

**Enumerate the complications of the peptic ulcers and discuss its management in brief.**

**Discuss the management of a patient with perforated peptic ulcer.**

**Ans. Modified Johnson Peptic ulcer classification**

Type	Location	Acid level	Vagotomy
1	Lesser curvature (m.c.)	Low	No role
2	Body of stomach and duodenum	High	Useful
3	Prepyloric antrum	High	Useful
4	High on lesser curvature, near GEJ	Low	No role
5	Anywhere but medication induced	Low	No role

**Features and complications**

Gastric ulcer	Duodenal ulcer
1. Pain after taking food and relieved by vomiting 2. Hematemesis common 3. Loss of weight seen 4. Spoke wheel pattern/hampton's line/penetrating sign/preserved peristalsis seen in benign gastric ulcer	1. Pain on empty stomach and relieved after eating 2. Melena common 3. Weight gain seen 4. Trifoliate duodenum or absent duodenal cap seen on barium study
Complications	
5. <b>Perforation into lesser sac</b> is the most common complication 6. <b>Malignant transformation</b> can occur 7. <b>Gastric outlet obstruction</b> can occur	5. <b>Bleeding</b> of gastroduodenal artery erosion into posterior wall is most common 6. Never occurs 7. <b>Gastric outlet obstruction</b> can occur 8. <b>Perforation</b> occurs in anterior wall

**Surgical options for peptic ulcer patients**

**Indications** have diminished since the advent of *H. pylori* therapy.

**Goal** – Decrease acid secretion from stomach.

**Options**

- Remove vagal stimulation via vagotomy
- Remove gastrin-driven secretion by performing an antrectomy
- Vagotomy with antrectomy is additive and decreases acid output by 85%.

**Procedures**

Vagotomy	Drainage (done with truncal vagotomy)	Resection and reconstruction	Type 4 gastric ulcer
Truncal vagotomy (above celiac and hepatic branch)	Pyloroplasty <ul style="list-style-type: none"> <li>• Heinke Mikulicz</li> <li>• Finney</li> <li>• Jaboulay</li> <li>• Weinberg modification of Heinke Mickulicz</li> </ul>	Subtotal gastrectomy	Kelling Madlener procedure for unstable patients
Selective vagotomy (below celiac and hepatic branch)	Gastroduodenostomy (Billroth 1)	Antrectomy	Csendes procedure for stable patients
Highly selective vagotomy	Gastrojejunostomy (Billroth 2)		Pouchet procedure
Hill baker procedure	Braun gastrojejunostomy		Shoemaker procedure
Taylor procedure	Roux-en-y uncut GJ		

**Truncal Vagotomy**

- Performed by division of the left and right vagus nerves above the hepatic and celiac branches just above the GE junction
- Some form of drainage procedure is used in association with truncal vagotomy
- Bile reflux may be more common after gastroenterostomy and diarrhea is more common after pyloroplasty. The incidence of dumping is the same for both.

**Highly selective vagotomy (Parietal Cell Vagotomy or proximal gastric vagotomy)**

- Divides only the vagus nerves supplying the acid-producing portion of the stomach within the corpus and fundus and preserves the vagal innervation of the gastric antrum so that there is no need for routine drainage procedures
- The criminal nerve of Grassi represents a very proximal branch of the posterior trunk of the vagus and great attention needs to be taken to avoid missing this branch in the division process as it can lead to ulcer recurrence if left intact.

**Truncal Vagotomy and Antrectomy**

- Antrectomy requires reconstruction of GI continuity that can be accomplished by a gastroduodenostomy (Billroth I procedure) or gastrojejunostomy (Billroth II procedure) or polya gastrectomy or Roux-en-Y gastrojejunostomy
- The retrocolic anastomosis minimizes the length of the afferent limb and decreases the likelihood of twisting or kinking that could lead to afferent loop obstruction.

Operation	Mortality (%)	Side effects (%)	Recurrence (%)
Vagotomy and antrectomy	2	5	1
Vagotomy and drainage	1	5	10
Highly selective vagotomy	0.2	1	10

**Gastric ulcers**

<b>Elective/Intractable cases</b>	Type 1	Distal gastrectomy and reconstitution
	Type 2 and 3	Truncal vagotomy and antrectomy
	Type 4	As mentioned above
<b>Perforation</b>	Stable Unstable	As above Patch closure after biopsy
<b>Bleeding</b>	Stable Unstable	As above Oversew and biopsy
<b>Obstruction</b>	Stable Unstable	As above Bypass and biopsy

**Duodenal Ulcers**

<b>Perforation</b>	Stable Unstable	Patch repair with truncal vagotomy (TV) patch closure
<b>Intractability</b>		Highly selective vagotomy
<b>Bleeding</b>	Stable Unstable	TV with pyloroplasty Oversew.
<b>Obstruction</b>	Stable Unstable	TV with antrectomy or GJ Bypass

**Management of perforated peptic ulcers is now described in the SN as follows:**

- Gastric ulcers perforate into lesser sac and duodenal ulcers perforate into pancreas through its anterior wall
- Clinical features of acute abdomen
- Patient present in shock and features of dehydration
- Patient also prefers lying still and has shallow respiration with diffuse abdominal pain and tenderness. Nausea, vomiting are also present
- **Important signs:** Generalized rebound tenderness/Guarding and obliteration of liver dullness suggest perforation peritonitis
- **Investigations:** Chest X-ray with both domes of diaphragm show free gas under diaphragm in 80% patients and is the only investigation required for diagnoses.
- **Management**
  - Resuscitation with isotonic fluids with two wide bore IV lines (green canula)
  - Antibiotics IV
  - Nasogastric aspiration, foley's catheterization and analgesia
  - Antisecretory agent infusion to be started
  - Rule out coagulopathy and monitor urine output and vitals of the patient
  - Arrange blood and take care of cardiopulmonary status of the patient
- Lastly, write the surgical management options and the management as shown in table
- Factors affecting the outcome in patients with peptic ulcer perforation include:

**(Mn: DASH)**

- **Delay** from initial diagnoses to treatment
- **Age** of the patient

- **Site:** Gastric has poorer prognosis
- **Hypotension** at presentation has a poor prognoses.

**Q15. Classify postgastrectomy problems and explain the pathophysiology and management of dumping syndrome.**

**Ans.**

Due to metabolic problems	Gastric reservoir dysfunction	Related to vagotomy	Related to reconstruction
Anemia – iron, B <sub>12</sub> , Folate deficiency	Dumping – early/ late	Diarrhea	Reflux gastritis
Osteopenia		Gallstones	Afferent loop syndrome
Weight loss		Gastric stasis	Efferent loop syndrome
			Roux syndrome
			Jejunogastric intususception
	Retained antrum syndrome		

**Incidence** – 25% and only 1% has permanent disabling symptoms.

**Dumping syndrome**

Can be early (20 to 30 minutes after eating) or late (2 or 3 hours after a meal).

*Early dumping*

- More common
- More often with Billroth II reconstruction than with Billroth I gastrectomy/ vagotomy and drainage procedures
- Occurs because of the rapid passage of food of high osmolarity from the stomach into the small intestine
- The hypertonic food bolus induces a rapid shift of extracellular fluid into the intestinal lumen to achieve isotonicity
- This causes luminal distention and induces the autonomic responses and release of neurotransmitters like serotonin, neurotensin, bradykinin-like substances
- More GI symptoms and fewer cardiovascular effects
- GI symptoms include nausea and vomiting a sense of epigastric fullness, cramping abdominal pain and often explosive diarrhea
- The cardiovascular symptoms include palpitations, tachycardia, diaphoresis, fainting, dizziness, flushing and occasionally blurred vision.

*Late dumping*

- Cause is rapid gastric emptying however, specifically to carbohydrates being delivered rapidly into the proximal intestine
- This triggers the release of large amounts of insulin to control the rising blood sugar level
- This results in an overcompensation so that a profound hypoglycemia occurs in response to the insulin
- This activates the adrenal gland to release catecholamines, which results in diaphoresis, tremulousness, light-headedness, tachycardia and confusion.

**Management**

- Dietary measures are usually sufficient
- Avoiding foods containing large amounts of sugar.
- Frequent feeding of small meals rich in protein and fat.
- Separating liquids from solids during a meal.
- Long-acting octreotide agonists have ameliorated symptoms
- Operative procedures include conversion of Billroth 2 to Billroth 1 or conversion of both to roux-en-y gastrojejunostomy, antiperistaltic jejunal loop placement to delay transit are some of the options to treat dumping syndrome.

**Q16. Discuss the causes of upper GI bleeding. Outline the management of a patient with upper GI bleeding.**

**Ans.**

**Upper GI bleed**

- Bleeding proximal to ligament of treitz is called upper GI bleed.

**Causes**

Nonvariceal (80%)	Portal hypertensive bleed (20%)
<b>Peptic ulcer (50-60%) m.c.</b>	Gastroesophageal varices (>90%)
Mallory weiss tear	Hypertensive gastropathy
Gastritis/duodenitis	Isolated gastric varices
Esophagitis	
AV malformations	
Tumors	
Angiodysplasia, hemobilia	

**Management***Step 1*

- Fluid and electrolyte Resuscitation
- Nasogastric decompression
- Rule out coagulopathy, Hematocrit, arrange blood
- Start IV antibiotic and infusion of proton pump inhibitors
- Rule out use of chronic NSAIDS, alcohol, smoking and steroids.

*Step 2*

In a stable patient, endoscopy to be done within 24 hours

Further management depends on endoscopic findings.

**1. Peptic ulcer bleed**

- Control endoscopically by using **4 quadrant (>13 ml total volume) epinephrine 1 : 10000 injection, electrocoagulation, argon plasma coagulation.**
- Duodenal ulcer bleeds more commonly, however, ulcer > 2 cm, gastric ulcers rebleed more commonly
- Once bleeding is controlled and patient gets stabilized, start medical management with Proton pump inhibitors and *H. pylori* eradication therapy

- **Indications of surgery in a case with peptic ulcer (gastrotomy with oversewing the blood vessel +/- vagotomy/total gastrectomy) include:**

- Failure to do endoscopy due to massive hemorrhage/hemodynamic instability
- Fail to control bleeding endoscopically
- Complications of endoscopy
- Rebleeding after controlling bleeding twice by endoscopy

## 2. Variceal bleed

- Manage as mentioned in the question on variceal bleed management.

## 3. Mallor weiss tear

- 80% resolve spontaneously
- If not, treat with
  - Local endoscopic therapy with epinephrine injection or multipolar electrocoagulation, endoscopic band ligation or endoscopic hemocliping
  - Angiographic intra-arterial infusion of vasopressin or transcatheter embolization, usually with an absorbable material such as a gelatin sponge done when endoscopy fails
  - High anterior gastrotomy and suturing of the mucosal tear with deep 2-0 silk ligatures to reapproximate the gastric mucosa is done when nonoperative techniques fail

4. **Other causes** are rare and managed according to the etiology. However, in any of these conditions, if the patient is unstable, proceed directly to surgery.

# SMALL INTESTINE

**Q17. Enumerate the causes of short bowel syndrome and discuss its management.**

**Write a note on intestinal failure.**

**Discuss in brief : surgical options to manage short bowel syndrome.**

**Ans. Short bowel syndrome is also called** Type 3 intestinal failure.

**Intestinal failure** – Inability to maintain protein – energy, fluid-electrolyte and micronutrient balance due to obstruction, dysmotility, surgical resection, congenital defect or disease associated absorption defect

**Hope hospital classification**

Type	Timing of use of TPN	Causes
1.	Short term (< 14 days)	Often perioperative ileus, pancreatitis, pseudo-obstruction, radiation enteritis, IBD
2.	> 28 days TPN need	Anastomotic leaks, enteroatmospheric fistulas, diseases requiring extensive enterectomy (volvulus, mesenteric ischemia)
3. (Short Bowel Syndrome)	> 6 months TPN need	Massive bowel resection due to various causes is irreversible



**Important causes of SBS**

Adult	Children
Mesenteric ischemia (m.c.) Crohn's disease Trauma abdomen Volvulus Motility disorders Desmoids	Gastroschises (m.c.) Volvulus Necrotizing enterocolitis Intestinal atresia Microvillus inclusion disease

**Short Bowel Syndrome** is defined as small intestinal length less than 200 cm or less than 30% of its prior normal length in that patient. This is an arbitrary value and the actual limit depends on the remnant bowel type as follows:

Type of SBS	Length that leads to SBS
1. Jejunostomy	<100–150 cm
2. Jejunocolic anastomosis	<60 cm
3. Jejunoleocolonic	<35 cm

**Pathophysiology**

Loss of ileocecal valve	Bacterial overgrowth
Loss of ileum	Bile salt and vitamin B12 deficiency
Loss of jejunum	Decreased nutrient absorption
Loss of colon	Decreased short chain fatty acid absorption Decreased carbohydrate fermentation

**Adaptation to SBS**

- Hyperphagia
- Increase in the absorptive surface area of intestine (structural adaptation)
- Alteration in gastrointestinal transit and motility (functional adaptation)

The adaptation can continue for up to 24 months.

**Management measures***Medical options*

- Fluid, electrolyte and acid-base balance
  - Parenteral support with enteral nutrition as tolerated
  - Oral rehydration and antimotility agents (loperamide, codeine, cholestyramine)
  - Octreotide is not to be used if the loss is < 3 litres as it decreases protein synthesis in the intestine and may potentially inhibit the adaptation process
    - Glucagon like peptide-2 analogues (**Teduglutide**) – enhances proliferative indices of adaptation by increasing crypt cell proliferation and villous height and increased expression of glucose transporters.
- Effect intestinal wet weight absorption by increase of around 1 liter/day and about 20% reduction in TPN requirement.

*Surgical options*

- **Autologous gastrointestinal reconstruction (AuGIR):** Aims to optimize the absorptive surface and function of remnant bowel by nontransplant procedures as follows:

- **SRSB** – Segmental reversal of small bowel – 10 to 12 cm jejunum is reversed and reanastomosed
- **Colonic interposition**
- **Intestinal lengthening procedures**
  - **STEP (Kim)** – serial transverse enteroplasty
  - **LILT (Bianchi)** – longitudinal intestinal lengthening and tailoring
- Both the procedures cause around 40% reduction in TPN requirement
- When all these procedures are unsuccessful, final step is to check for indications of intestinal transplant and enrol the patient.

**Indications for small bowel transplant are as follows:**

- SBS with overt or impending liver failure caused by TPN induced liver disease
- SBS with multiple central line thrombosis
- > 2 episodes of central line infection or hospitalization or single evidence of fungal infection
- Frequent severe dehydration despite IV fluid supplementation and TPN.

**Q18. Write the causes of Enterocutaneous fistula.**

**Write a note on management of a patient with enteroatmospheric fistula.**

**Discuss GI – cutaneous fistulas.**

**Enumerate the factors that interfere with spontaneous closure of the enterocutaneous fistulas.**

**Ans.**

- Fistula word is latin meaning pipe/flute
- It is defined as an abnormal communication between two epithelialized surfaces
- **M.C. cause** – iatrogenic
- **Types**

Based on communication	Internal External (enterocutaneous)
Based on output	High output (> 500 ml/day) Moderate output Low output (< 200 ml/day)

- **Webster and Carrey classification of causes of small intestinal fistulas**
  - TYPE A: Congenital (malformations)
  - TYPE B: Trauma related or damage control surgery related
  - TYPE C: Infective etiology (actinomycosis, tuberculosis, cytomegalovirus, etc.)
  - TYPE D: Irradiation enteritis, inflammatory cause (Crohn's, ulcerative colitis, diverticulitis), Tumor (small bowel, large bowel malignancies)
  - TYPE E: Perforation with abscess. Includes iatrogenic injuries or traumatic injuries when associated with abscess.

**Common age related causes of colovesical fistula**

- Crohn's (20 to 40 yrs age group)
- Colorectal malignancy (40 to 60 yrs age group)
- Diverticulitis (50 to 80 yrs age group, **overall m.c. cause**)

**Management outline of enterocutaneous fistulas is as follows:**

No.	Phase	Days from Diagnoses	Interventions
A	Stabilization	1–2 days	<ul style="list-style-type: none"> <li>Fluid, electrolyte, acid—base balance</li> <li>Nutrition enteral or parenteral and immune enhancing formulas with care to avoid refeeding syndrome</li> </ul>
			<ul style="list-style-type: none"> <li>Control of infection</li> <li>Clearance of dead space/collections</li> <li>Skin care measures (sump suction, NVAC device) and avoidance of bed sores and DVT in bedridden patients</li> <li>Control of output (proton pump inhibitors, octreotide, infliximab)</li> </ul>
B	Investigation	7–10 days	<ul style="list-style-type: none"> <li>Fistulogram</li> <li>CECT for intraabdominal collection/dead spaces</li> <li>Colonoscopy/UGI endoscopy as indicated</li> </ul>
C	Decision	Upto 4–6 weeks	<ul style="list-style-type: none"> <li>Wait and watch approach</li> <li>Proceed to definitive surgery if there is presence of any of the following (Mn: FRIENDS—<b>factors affecting spontaneous fistula closure</b>)               <ul style="list-style-type: none"> <li>Foreign body (suture, mesh, debris)</li> <li>Radiation as etiology</li> <li>Inflammation/infection</li> <li>Epithelialization with tract length &lt; 2.5 cm long with &gt; 1 cm<sup>2</sup> defect in bowel wall</li> <li>Neoplasm</li> <li>Distal obstruction</li> <li>Steroids therapy</li> <li>High output (&gt; 500 ml/day)</li> </ul> </li> <li>Planning of operative approach</li> <li>Optimal time for operation</li> </ul>
D	Definitive surgery	After 2 months or interim intervention in inevitable circumstances	<ul style="list-style-type: none"> <li>Resection and anastomosis with or without feeding tubes (gastrostomy/jejunostomy)</li> <li>Diversion stoma followed by restoration of intestinal continuity at a later date</li> </ul>
E	Healing and Rehabilitation	Ongoing phase after A-D	<ul style="list-style-type: none"> <li>Continue nutritional support</li> <li>Physical and mental rehabilitation</li> <li>Complete oral/enteral diet</li> </ul>

**Q19. Write a note on Meckel's diverticulum.**

**What is Meckel's diverticulum? Write its clinical features. Discuss the management of an incidentally discovered Meckel's diverticulum during Surgery.**

**Ans. Introduction**

- Most common congenital anomaly of the small intestine

- Occur in 2% of the population
- Equal incidence in men and women
- It is located on the antimesenteric border of the ileum 45 to 60 cm proximal to the ileocecal valve.

### **Etiology**

Results from incomplete closure of the omphalomesenteric or vitelline, duct.

This failure can result in any of the following manifestations:

- Omphalomesenteric ligament (fibrous band)
- Omphalomesenteric fistula
- Omphalomesenteric cyst
- Meckel's diverticulum.

*Heterotopic tissue within the Meckel diverticulum*

- Gastric mucosa (**M.C.**)
- Pancreatic mucosa is encountered in about 5% of diverticula
- Colonic mucosa.

### **Clinical Manifestations**

- Incidental finding during autopsy or surgery
- Most common clinical presentation in children is gastrointestinal bleeding
- Most common clinical presentation in adults is intestinal obstruction which can be due to
  - Volvulus around fibrous band which connects meckel's to umbilicus
  - Intussusception with meckel's as a lead point
  - Incarceration of the diverticulum in an inguinal hernia (**Littre's hernia**)
- Diverticulitis—more common in adult patients and is clinically indistinguishable from appendicitis
- Benign tumors—Leiomyomas, angiomas, and lipomas
- Malignant tumors—Adenocarcinomas, which generally originate from the gastric mucosa, sarcoma and carcinoid tumor.

### **Diagnoses**

- Scintigraphy with sodium 99 mTc-pertechnetate  
More accurate in children than in adults where, the sensitivity and specificity can be improved by the use of pentagastrin and glucagon or histamine 2 (H2) receptor antagonists (e.g. cimetidine)
- In adult patients, when nuclear medicine findings are normal, barium studies should be performed
- In patients with acute hemorrhage, angiography is sometimes useful.

### **Treatment**

- **Symptomatic Meckel's diverticulum**—Open or laparoscopic diverticulectomy or segmental ileal resection which is required for treatment of patients with bleeding because the bleeding site is usually in the ileum adjacent to the diverticulum caused by ulceration due to the acid secretion by the heterotopic gastric mucosa
- **Asymptomatic diverticula**—Should be resected in all patients upto age of 80 years if patient can tolerate the extra time required in the procedure because a 6.4% rate of

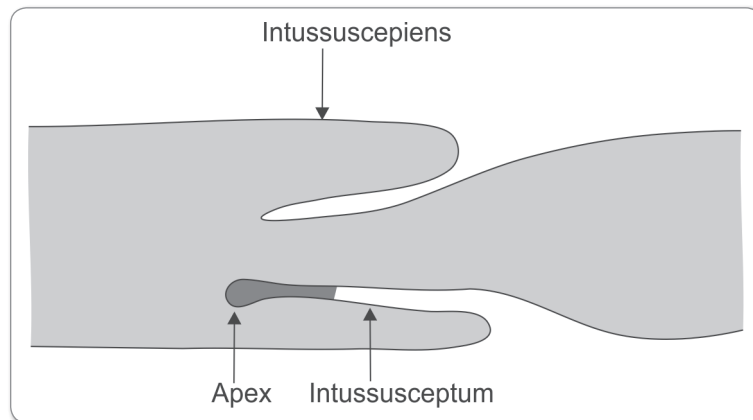
development of complications from the Meckel's diverticulum is calculated to occur over a lifetime whereas the morbidity of the procedure is only around 2%.

**Q20. What is Intussusception? Discuss its causes and management.**

**Ans.** Intussusception is telescoping of one portion of the intestine into the other.

**Causes**

- Idiopathic (m.c.)
- Peyer patch hypertrophy (viral gastroenteritis, URI, administration of rotavirus vaccine) (3 months to 3 years)
- Meckel's diverticulum (1 year onward)
- Malignancy of bowel or mesentery
- Intestinal polyps (Peutz Jegher's syndrome), Intestinal duplication
- Inflamed appendix
- Submucosal lipoma
- Submucosal hemorrhage associated with Henoch-Schönlein purpura
- Foreign body
- Ectopic pancreatic or gastric tissue
- Postoperative small bowel intussusception in the absence of a lead point.



**Fig. 6:** Intussusception

**Clinical Presentation**

- Severe cramping abdominal pain, vomiting, passage of bloody mucous (red currant jelly stool)
- Examination reveals a **sausage shaped mass** in the abdomen which may sometimes resolve spontaneously and is therefore an example of 'vanishing lump'
- Emptiness in the right iliac fossa in cases is called **Sign of Dance**
- The child often draws the legs up during the pain episodes and is usually quiet during the intervening periods.

**Diagnoses**

- Abdominal ultrasound - **target sign** on a transverse view and **pseudokidney sign** when seen longitudinally.

**Treatment***Nonoperative Management*

- Hydrostatic reduction by enema using contrast/air/barium
- **Contraindications** - Presence of peritonitis or hemodynamic instability
- Successful reduction is accomplished in more than 80% of cases and confirmed by resolution of the mass along with reflux of air into the terminal ileum
- Recurrence rate - 11% usually managed by another hydrostatic reduction. A third recurrence is an indication for operative management.

*Surgical Management*

- **Indications**
  - Presence of peritonitis
  - Failed hydrostatic reduction
  - Third recurrence
  - Complete small bowel obstruction
- The surgical reduction is done in a retrograde fashion
- Bowel resection is required in cases in which the intussusception cannot be reduced, the viability of the bowel is uncertain, and/or a lead point is identified
- An ileocolectomy with primary reanastomosis is usually performed. An appendectomy is also performed simultaneously
- It can also be done using laparoscopy.

**Q21. Give a list of causes of neonatal intestinal obstruction and discuss management in brief.**

**Write a note on necrotizing enterocolitis.**

**Write a note on meconium ileus.**

**Discuss the causes and management of a newborn presenting with intestinal obstruction.**

**Ans.**

Proximal	Distal
<ul style="list-style-type: none"> <li>• Duodenal atresia</li> <li>• Malrotation</li> <li>• Midgut volvulus</li> <li>• Annular pancreas</li> <li>• Pseudoportal vein</li> </ul>	<ul style="list-style-type: none"> <li>• Jejunal atresia &gt; ileal atresia</li> <li>• Meconium ileus</li> <li>• Hirschsprung's disease</li> <li>• Meconium plug syndrome</li> <li>• Necrotizing enterocolitis</li> <li>• Colonic atresia</li> </ul>

**Duodenal atresia**

- **Types**
  - Mucosal stenosis
  - Mucosal web with normal muscular layers (Windsock deformity)
  - Two ends joined by fibrous cord
  - Two ends separate
- Characteristic radiological appearance—Double bubble sign
- Association with down syndrome

- **Management**

**Diamond shaped duodenoduodenostomy with/out tapering duodenoplasty** done by giving incision transverse in the bowel proximal to atresia and longitudinal in distal bowel and anastomosing them.

**Jejunioileal atresia**

- Types
  - Mucosal web
  - Fibrous cord
  - A – V- shaped small mesenteric defect
  - B – apple peel/Christmas tree large defect
  - String of beads/string of sausage appearance due to multiple small atretic segments in between normal segments
- Type 3 B and 4 have a retrograde blood supply
- Characteristic radiologic sign – Triple bubble sign
- **Management** – Multiple anastomosis over stent or resection anastomosis with or without a tapering enteroplasty.

**Malrotation**

- **Normal gut rotation** is 270°C counterclockwise.
- **Normal Ladd bands** – Go from cecum and an ascending colon to retroperitoneum and lateral abdominal wall
- Normal SMV and SMA relation—SMV is on the right side of SMA
- Midgut volvulus—rotates clockwise
- Surgery—Do counterclockwise rotation to correct the problem
- **Most common rotation abnormality is Non rotation**
- **Most common malrotation—incomplete rotation**
- Other rotation abnormalities—partial rotation and reverse rotation
- In malrotation, SMV comes to left of SMA because of '**whirlpool sign of malrotation** appreciated on CECT with CT angiogram – SMV with mesentery rotated around the axis of SMA'
- Other signs – **corkscrew duodenum**
- Duodenojejunal flexure appearing in upper GI barium study before the pedicle of L2 vertebrae
- **Ladd Procedure** – Main steps include:
  - Widen the base of mesentery
  - Relieve volvulus
  - Appendectomy.

**Necrotizing enterocolitis**

- **Most common GI surgical emergency in neonates**
- **Most common site involved is ileal mucosa**
- Very important cause of short bowel syndrome in children
- Prematurity is the most important risk factor for developing necrotizing enterocolitis
- Patients with ARDS are also at increased risk

- Pneumatosis intestinalis that develops here contains hydrogen
- **BELL'S Staging system is used to stage necrotizing enterocolitis**
- **Management**
  - Conservative management is successful in 90% cases.
  - Resuscitation
  - Look for ominous signs and indications for surgery and if present immediate surgery is life saving.

#### *Indications for surgery*

- Fixed palpable and visible bowel loops with abdominal distension
- Nonresponder to medical therapy
- Resistant thrombocytopenia
- Signs of perforation peritonitis
- Progressive acidoses
- Erythema and edema of bowel wall.

However, it should be remembered that surgery does not prevent disease progression. It is the management of general condition that saves the life here.

**m.c. site of stricture after necrotizing enterocolitis – splenic flexure.**

#### **Meconium Ileus**

- **Triad of** generalized abdominal distension, bilious vomiting and non passage of meconium for 12 to 24 hours after birth.
- Usually a sign of presence of cystic fibroses (can be confirmed with pilocarpine iontophoresis test). Other obstructing condition associated with cystic fibroses is **jejunoileal atresia**
- Abdominal X-ray features – snowstorm appearance
- Complicated meconium ileus – meconium ileus with perforation

**Management**—hydrostatic gastrograffin contrast enema and 5 ml 10% N acetylcysteine orally 6 hrly.

If the patient does not resolve by this conservative means, treatment is enterotomy f/b 4% N acetylcysteine with warm saline or enterotomy and pushing the contents into colon f/b closure of enterotomy.

#### **Meconium plug syndrome**

It is not associated with meconium ileus. It is a large intestinal obstruction associated with

- Hirschsprung disease
- Maternal diabetes
- Hypothyroidism

#### **Q22. Enumerate the causes of mesenteric ischemia.**

**What are the types of mesenteric ischemia? Discuss its management in brief.**

**Write a note on intestinal angina.**

**Discuss acute mesenteric ischemia.**

**Ans.** Mesenteric ischemia can be acute or chronic



**Acute mesenteric ischemia***Causes*

Occlusive	Increased wall tension	Non occlusive	Inadequate outflow
Embolic (m.c.)	Closed loop obstruction	Cardiogenic shock	Mesenteric venous thrombosis
Thrombus	Pseudoobstruction	Hemorrhagic shock	Mesenteric nodal disease
Volvulus		Septic shock	Pancreatic neoplasms
Strangulated hernia		Critically ill patients	
Aortic dissection		Pancreatitis	
Aortic insufficiency		Burns	
Mesenteric tear			

*Acute superior mesenteric ischemia*

- Both sexes are affected equally
- Embolic cause (myocardial infarction, cardiac thrombus, atrial fibrillation) > thrombotic cause (cerebral infarction, PVD, coronary artery disease, disseminated cancer).
- Mesenteric venous thrombosis causes include thrombophilia, OC pills, liver cirrhoses and inflammatory bowel disease.

*Clinical features*

- Symptoms are out of proportion of physical signs
- Any patient with sudden onset pain abdomen > 2 hours with no other definite cause should be evaluated for AMI
- All causes of acute abdomen are included in its differential diagnoses.

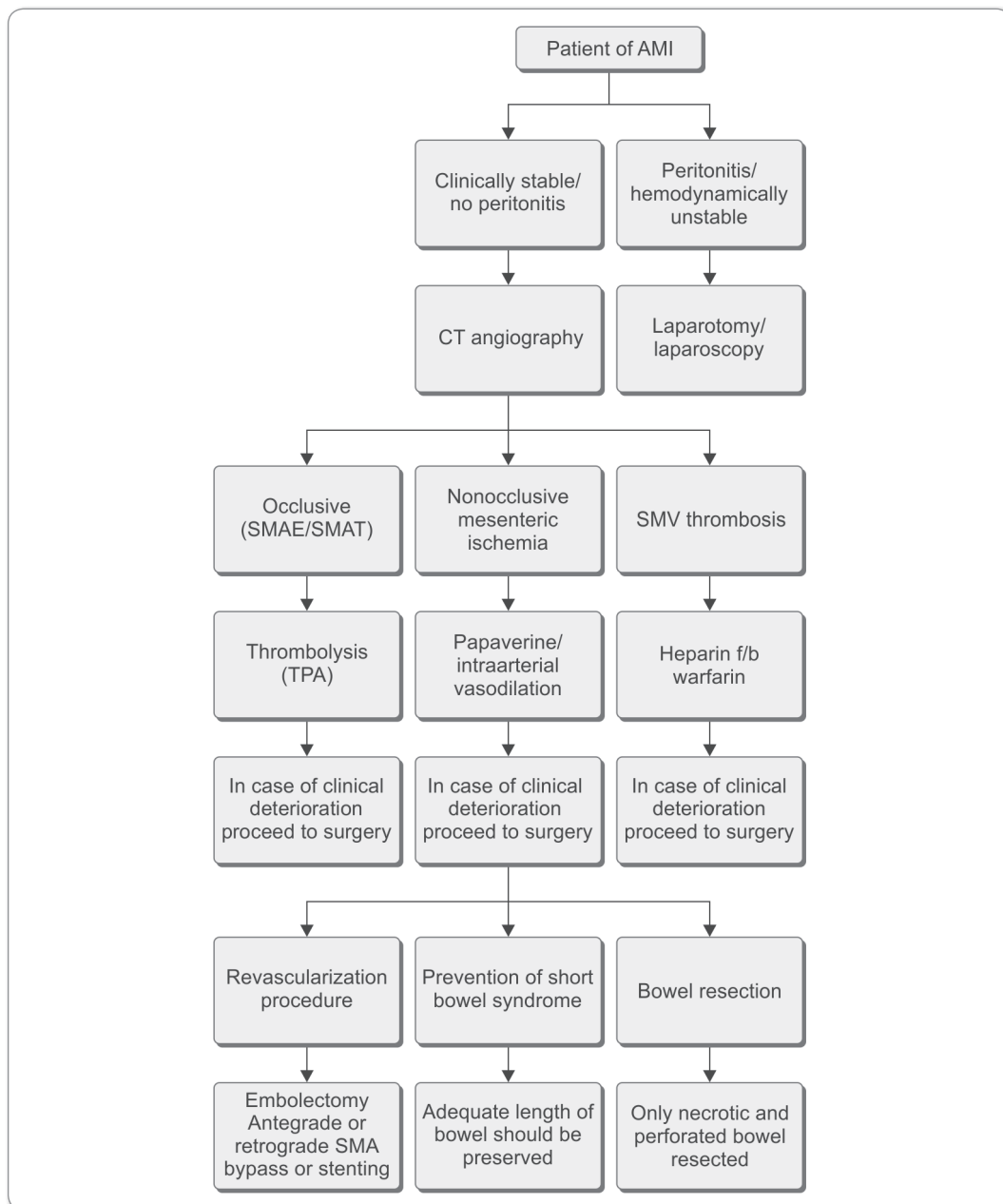
*Investigations*

- Increased TLC, metabolic acidoses, hyperamylasemia, elevated lactate levels are seen.
- Sensitive and specific marker is alfa-glutathione-s- transferase
- X-ray abdomen—dilated loops, pneumatosis intestinalis and pneumatosis portalis may be seen
- Investigation of choice for arterial occlusion is CT angiography.
- Others—mesenteric angiography – diagnostic and therapeutic and gold standard
- MR angiography
- Duplex scanning of mesenteric vessels
- Diagnostic laparoscopy.

*Management*

Resuscitation of patient with oxygen, IV fluids, electrolyte correction, pain relief and antibiotics.

- Primary anastomosis of bowel should not be done if revascularisation is carried out
- Intraoperative determination of cause of mesenteric ischemia



**Mortality:** NOMI > SMAT > SMAE > SMVT

### Chronic mesenteric ischemia

- Most common cause—atherosclerosis

- Other causes—vasculitis, takayasu disease, SMVT
- It is more lethal than acute mesenteric ischemia because if SMAT complicates a case of chronic mesenteric ischemia, it has a very high mortality rate.

#### *Clinical features*

- Old age, females
- **Mesenteric angina** (postprandial colicky epigastric pain)
- Weight loss

**Site:** Celiac > IMA > SMA

**Investigations:** same as above

#### *Management*

- Revascularisation is indicated in symptomatic disease
- Short, nonostial, focal disease—endovascular angioplasty
- 2 or more than 2 vessels with critical stenosis—open SMA revascularisation with antegrade or retrograde bypass (retrograde means through infra renal aorta or iliac vessel)

#### *Results*

- Mesenteric angioplasty and stenting has high success rate with low morbidity and mortality
- Open bypass on the other hand has decreased rate of restenosis and decreased incidence of symptomatic recurrences.

### **Q23. Write a note on superior mesenteric artery syndrome.**

**Ans.**

**(Imp: Always Remember:** The angle between abdominal aorta and superior mesenteric artery is normally acute (40 to 60 degrees), when it becomes more acute (5 to 25 degrees), it causes compression of the third part of duodenum which results in SMA syndrome.

On the other hand, the compression of left renal vein between abdominal aorta and Superior mesenteric artery is known as nutcracker syndrome.

- Superior mesenteric artery syndrome is also known as Wilkie syndrome, Cast syndrome, mesenteric root syndrome or arteriomesenteric duodenal ileus.
- It is the compression of the third part of the duodenum between Aorta and SMA.
- **Established dimensions to label SMA syndrome**
  - **Aorta – SMA angle** < or = 25° (Normal – 40 to 60°)
  - **Aortomesenteric distance** < 9 mm (Normal – 10 to 20 mm)

#### **Etiopathogenesis**

- Any factor that decreases the retroperitoneal fat pad and lymphatic tissue decreases the protection of third part of duodenum from SMA and predisposes to SMA syndrome
- It can be **chronic relapsing congenital** variant or **acute/induced variant**
- **Predisposing factors**
  - Recent rapid weight loss
  - Prolonged supine immobilization
  - Tall asthenic women

- Scolioses
- Placement of body cast
- After left nephrectomy surgery.

#### *Clinical presentation*

- Chronic congenital variant patients have a lifelong history of intermittent exacerbations of symptoms such as nausea, vomiting, weight loss, epigastric pain especially after eating
- Acute variant can present with above symptoms with a sudden and unremitting nature
- Patients can also present in shock, Upper GI bleeding, Perforation peritonitis, dehydration and fluid and electrolyte abnormalities due to persistent vomiting.

#### *Diagnoses*

- CECT with CT angiography with lateral films
- Upper GI Barium study or hypotonic duodenography can also be used.

#### *Treatment*

- **Acute variant** – usually responds to conservative measures which include the following:
  - NPO and Nasogastric decompression
  - Resuscitation and correction of the fluid and electrolyte abnormalities
  - Monitoring of temperature, pulse, blood pressure and urine output
  - Metoclopramide or other prokinetic agent has also been tried.
  - Jejunostomy feeds or parenteral hyperalimentation.

**The goal of the conservative measures is restoration of weight which will take care of the symptoms.**

- **Chronic relapsing cases and cases which do not improve on the conservative management require surgery.**
- Duodenojejunostomy (Open/Laparoscopic, anatomic/Roux-en-Y) is the operative procedure of choice.

#### **Q24. What is morbid obesity? Discuss its management in brief.**

**Give the classes of obesity for Indian population. Discuss the etiopathogenesis and management of morbid obesity in current scenario.**

**Ans.**

BMI classes	Asia	Others
Underweight	–	<18.5
Normal	<23	18.5–24.9
Overweight	23–24.9	25–29.9
Mild obesity (1)	25–32.4	30–34.9
Severe obesity (2)	32.5–37.4	35–39.9
Morbid obesity (3)	>37.5	40–49.9
Superobese	–	> 50 kg/m <sup>2</sup>

#### **Definition**

BMI > 40 kg/m<sup>2</sup> or twice the ideal body weight for that age and height.

**Pathophysiology**

- Familial predisposition
- Increased levels of ghrelin cause increased food intake
- Metabolic syndrome—Type 2 diabetes, impaired glucose tolerance, hypertension, dyslipidemia

**Medical therapy**

- 10% weight loss at the rate of 0.5 to 2 lb/ week
- Maintenance of weight loss for 6 months
- Plan is to give very low calorie diet with decreased fat intake and decreased carbohydrate intake
- **Drugs approved** – Sibutramine (SNRI—serotonin and norepinephrine reuptake inhibitor) and Orlistat (pancreatic lipase inhibitor)

**Indications for surgery**

- BMI > 40 kg/m<sup>2</sup>
- BMI >35 kg/m<sup>2</sup> with co-morbidity
- With failed medical therapy, motivated attitude and psychiatric stability
- Knowledgeable about operation
- No medical problems precluding survival.

**Absolute contraindication** - Prader Willi syndrome

**Preoperative evaluation**

- Documented medically supervised diet
- First generation cephalosporin for 24 hrs preoperation
- Ultrasound for gallstones
- UGI endoscopy for GERD
- Orthopedic, psychiatric, nutritionist, physician and endocrinologists consult
- To prevent DVT – sequential compression device boots, ambulation within 4 to 6 hrs and LMWH for 2 weeks
- ABGA.

**Operations***Restrictive*

- Vertical banded gastroplasty
- Adjustable gastric banding (Fielding and Allen)
- Sleeve gastrectomy
  - Has superior weight loss and better appetite control than RYGB.
  - Advantages—technical simplicity, no malabsorption or internal hernias, no need for serial readjustments
  - Disadvantage—leak, bleeding

**Mildly malabsorptive**

- Roux-en-y gastric bypass

**Largely malabsorptive**

- Biliopancreatic diversion
  - Most effective of these procedures
- Duodenal switch

Estimated weight loss is highest for BPD/DS – 70.1%

Long-term complications – BPD – protein malnutrition and RYGB – iron and vitamin B<sub>12</sub> deficiency.

All these procedures are now being done using laparoscopy and even robotic surgery which has been found to be more beneficial in the superobese group of the patients.

**Q25. Classify abdominal tuberculosis and discuss its management.****Write a note on gastrointestinal tuberculosis.**

**Ans. Most common type of abdominal TB—peritoneal TB**

**Most common site in intestinal TB—ileocecal region**

abdominal tuberculosis can be classified as follows:

Solid visceral TB	Liver, spleen, pancreas, bile duct	
Intestinal	Ulcerative -----	Deep and transverse ulcers
	Hyperplastic/hypertrophic Structuring/sclerotic/fibrotic -----	Napkin ring strictures
Peritoneal	Wet (ascitic) -----	Localized/encysted/generalized
	Dry -----	Adhesive/plastic/miliary
	Omentum -----	Rolled up omentum
	Mesenteric lymph nodes -----	Mesenteric adenitis/abscess/tubercles mesenterica
	Adhesions / mesentery involvement -----	Mesenteric cyst Adhesive obstruction

**Pathogenesis**

- Primary—From contaminated food (*Mycobacterium bovis*)
- Secondary—Due to swallowed sputum containing *M. tubercle bacillus*
- Earliest pathological changes occur in submucosa of the intestine.
- Ulcers are transversely placed in TB and longitudinally placed in typhoid
- The **ulcerative form** occurs when the microbe has a stronger hand than the host immune response. The **hyperplastic form** occurs when the host immune response is stronger than the organism.

**Clinical features**

- Nonspecific abdominal pain (**m.c.**)
- Fever, night sweats, anorexia, weight loss.
- Can present as recurrent episodes of subacute intestinal obstruction
- Can also present as malabsorption, bowel perforation and GI hemorrhage.

**Investigations**

- TLC – lymphocytosis

- Raised ESR (**m.c.**)
- Ascitic fluid
  - Coagulum on standing
  - ADA > 42 (less specific in presence of HIV, cirrhoses and malignancy. Otherwise 90 to 95% sensitive and specific)
  - Protein > 2.5
  - SAAG < 1.1
  - Lymphocytosis > 500 mm<sup>3</sup>
  - AFB by ZN stain (< 5% chance of being positive)
  - AFB by culture of ascitic fluid – positive in 20 to 45% cases

Yield of AFB is low in extrapulmonary TB, still it is comparatively more in ascitic/miliary TB greater than in intestinal/solid visceral TB.

- **Barium meal follow through**
  - Thickening of ileocecal valve
  - Umbrella sign/fleishner sign
  - Pulled up cecum due to shortening of mesocolon
  - Narrowed terminal ileum (sterlin sign)
  - Flocculation of barium
  - Gooseneck deformity due to loss of ileocecal angle
  - String sign due to narrowing of terminal ileum
  - Sinus, fistula, stricture can be seen
- **Ultrasound abdomen**
  - Club sandwich appearance
  - Mesenteric thickening (> 15 mm)
- Laparoscopy is the single most important test in peritoneal TB
- **PCR – in any body fluid/biopsy has sensitivity/specificity/positive predictive value – 85/99/95%.**

### Management

- Conservative medical management with **DOTS** (Anti-tubercular medication) is the cornerstone of treatment
- Steroid is used in cases of ascetic TB as it is believed to decrease adhesion formation.

### Indications of surgery

- Complete obstruction/ strangulation
- Second episode of partial obstruction after starting ATT
- 1st episode of obstruction in patient on ATT for greater than 2 to 3 months
- Free intestinal perforation
- Severe intestinal hemorrhage
- Elective in failed medical therapy
- In cases of doubtful diagnoses to rule out malignancy
- Intraabdominal abscess/fistula (internal/external) due to contained perforation.

### Principle of surgery

- Removal of all adhesions/distal obstruction/strictures/fistulas which is called refunctionalization of bowel

- To not operate in a case from 10 days to 4 to 6 weeks after a contained perforation in TB (and also in cases after 1 surgery) as this is the phase of obliterative peritonitis due to fistula/peritoneal inflammation/previous surgery.

**Q26. What is intestinal obstruction? Enumerate the causes of intestinal obstruction.**

**Ans. Intestinal obstruction** refers to the partial or complete mechanical or non-mechanical blockage of the small or large intestine.

1. There are two types – **Dynamic** (when there is an actual mechanical cause) and **Adynamic** [no mechanical cause but occur due to absent paralysis (**Ileus**) or nonpropulsive peristalsis (**Pseudo-obstruction**)]
2. Other classification is as follows
  - **Acute obstruction:** Present with a sudden onset of the cardinal symptoms of intestinal obstruction.
  - **Chronic obstruction:** Usually seen in large bowel obstruction with lower abdominal colic and absolute constipation followed by distension.
  - **Acute on chronic obstruction:** There is a short history of distension and vomiting on a background of pain and constipation.
  - **Subacute obstruction:** Incomplete obstruction.
3. One other classification of intestinal obstruction is based on the severity of symptoms
  - **Simple obstruction:** Intact blood supply of the obstructed part
  - **Strangulated obstruction:** Blood supply is hampered.

**Causes of intestinal obstruction are as follows:**

*Dynamic*

- Intraluminal
  - Foreign bodies (coin, battery), food or fecal Impaction, Worm ball (ascaris), bezoars, gallstones
- Intramural
  - Stricture, malignancy
- Extramural
  - Adhesions, hernia, malignancy, volvulus, intussusception
  - Adynamic
- Paralytic ileus, mesenteric vascular occlusion, Pseudo-obstruction.

(**NOTE:** Causes can also be divided as neonatal and adult causes. Refer the question on neonatal intestinal obstruction for the list)

**Q. Discuss the clinical features of acute intestinal obstruction.**

**Ans.**

**Symptoms**

Cardinal symptoms in any case of acute obstruction are:

1. Abdominal pain—colicky with or without history of gola formation. Pain occurs around the umbilicus (small bowel) or hypogastrium (large bowel). In cases with long standing obstruction, colics are replaced by a constant dull aching pain.
2. Abdominal distension—Central in small bowel obstruction and flank fullness in large bowel obstruction



3. Nausea and vomiting—Early in case of small bowel obstruction and late in case of large bowel obstruction
4. Obstipation > constipation – early in case of large bowel obstruction and late in case of small bowel obstruction. Patients with small bowel obstruction may have episodes of diarrhea initially before progressing to obstipation and constipation as initially, the peristalsis is normal below the level of obstruction till it is empty of its contents when if the obstruction is not relieved, it collapses and shuts down its lumen.

**Exceptions (Obstruction without constipation)** include partial obstruction, Richter's hernia, mesenteric ischemia, gallstone ileus and obstruction secondary to pelvic abscess.

Other presenting forms alongwith these symptoms

- Abdominal lump suggests intussusception, malignancy, volvulus etc.
- Anorexia, weight loss and fever may point towards malignancy or tuberculosis
- Features of fever, diaphoresis, palpitations may suggest strangulation.

#### **Localizing based on symptoms**

- **In high small bowel obstruction**, vomiting occurs early with rapid dehydration. Distension is minimal.
- **In low small bowel obstruction**, pain is predominant with central distension. Vomiting is delayed.
- **In large bowel obstruction**, distension is early. Pain is mild and vomiting and dehydration are late.
- **In strangulation** – constant pain, abdominal tenderness and rigidity are important features.

#### **General physical examination**

- Tachycardia, tachypnea, elevated temperature, signs of dehydration and agitated state may suggest complete obstruction or strangulation
- Anemia, lymphadenopathy may be present.

#### **Inspection findings**

- Visible peristalsis, visible bowel loops, scars of previous surgery, hernia orifices
- Abdominal distension—central or peripheral as discussed above.
- Abdominal lump might be present.

#### **Palpation**

- Doughy feel of abdomen
- Palpable bowel loops
- Hernia might be present
- Tenderness, rebound tenderness, guarding and rigidity suggest peritonitis.

#### **Percussion and auscultation**

- Obliteration of liver dullness suggest perforation peritonitis.
- Increased frequency of bowel sounds in early obstruction (borborygmi) and decreased or absent bowel sounds in late obstruction, strangulation and perforation.

**Q. Discuss the management of a case of acute small bowel obstruction.**

**Q. Discuss the management of a patient with acute abdomen.**

**(The investigation and resuscitation part are the same in both the questions. Further management of acute abdomen depends on the cause of acute abdomen so not specified. Step 2 of answer is only for the management of a case of acute intestinal obstruction as a cause of acute abdomen).**

**Ans.**

### **Investigations**

(Radiological investigations are discussed in the radiology section)

### **Other investigations**

#### **• Blood investigations**

- Hematocrit, hemoglobin level, Total leucocyte counts, electrolytes (**Sodium, Potassium, Calcium, magnesium**) Platelet count and coagulation parameters, albumin levels, liver and kidney function, **sugar level**, amylase and lipase are the important blood investigations.
- These help in **ruling out the important causes** of acute presentation as small bowel obstruction/acute abdomen such as hypocalcemia, hypokalemia, hypomagnesemia, diabetic ketoacidosis, uremia, pancreatitis and adrenal insufficiency at the outset.
- Other important **medical conditions presenting as acute abdomen** include Porphyria (urine discoloration is characteristic), tabes dorsalis, herpes zoster at T 10 or so level, poisoning with lead and these should always be remembered while treating these patients.
- Always send blood for liver and kidney function, arterial blood gas analysis, coagulation profile and grouping and cross matching to arrange blood and assess fitness of the patient in case a surgery is required.
- Electrocardiogram, SpO<sub>2</sub> measurement is another important parameter before surgery.

### **Treatment outline**

#### *Step 1*

- Resuscitation is the most important step. Fluid balance, electrolyte management especially potassium and magnesium, correction of acid- base balance once the fluid balance is taken care of are the life saving measures before surgery
- Nasogastric tube placement for gastrointestinal decompression
- Monitoring of urine output, intake of fluids, pulse, blood pressure, temperature and respiratory rate is very important during this phase of resuscitation
- If necessary, a central venous line can be inserted to give fluid as per central venous pressure measurement
- Antibiotics are to be given if the patient has a closed loop obstruction or if the patient is in sepsis or if he is planned for operation
- The conservative measures as mentioned above can relieve cases of partial subacute obstruction and should be tried for 12 to 18 hours if possible.

*Step 2 (Role of surgery after adequate resuscitation)*

- Follow the dictum “the sun should not both rise and set on a case of unrelieved obstruction”
- **Indications for early surgery** without conservative trial of 12 to 16 hours
  - Closed loop obstruction
  - Strangulated obstruction
  - Internal or external obstructed or strangulated hernia/volvulus as the cause of obstruction
- **Principles in intestinal obstruction surgery**
  - Identify the segment that is obstructed and look for its viability and nature of obstruction
  - Identify the cause of obstruction
  - Operative decompression of the proximal dilated bowel to decrease tension on the site of obstruction and surgery.
- **Surgical Options once the site is identified include–**
  - Adhesiolysis of bands
  - Excision and stoma or reanastomosis
  - Bypass and reestablishment of continuity
  - Operative decompression
- **Treatment in cases of recurrent band obstruction**
  - Repeat adhesiolysis
  - Noble’s plication operation
  - Charles–Phillips transmesenteric plication
  - Intestinal intubation

Rest of the surgical management depends on the specific cause of obstruction.

**Q. Enumerate the causes of adynamic intestinal obstruction.**

**Ans.**

**Adynamic intestinal obstruction has three main causes:**

Ileus, Pseudo-obstruction and mesenteric vascular events. Mesenteric vascular events have already been discussed.

**Causes of paralytic ileus**

- Post – operative period after abdominal surgery
- Electrolyte abnormalities (hyponatremia, hypokalemia, hypomagnesemia, hypermagnesemia)
- Drugs (Opiates, anticholinergics, tricyclic antidepressants, calcium channel blockers)
- Endocrine disorders (Hypothyroidism, adrenal insufficiency)
- Uremia, mesenteric ischemia, myocardial infarction
- Retroperitoneal hemorrhage
- Infections (peritonitis, intra-abdominal abscess, pneumonia)
- Spinal shock patients

**Causes of intestinal pseudo-obstruction**

- Acute – Ogilvie's syndrome
- Primary variant – Visceral myopathies/GI motility disorders
- Secondary variant –
  - Endocrine disorders (hypothyroidism, diabetes, hyperparathyroidism)
  - Drugs (opiates, neuroleptics, anticholinergics)
  - Autoimmune (SLE, dermatomyositis, scleroderma)
  - Parkinson's disease, retroperitoneal hematomas.

## LARGE INTESTINE

**Q27. Write a note on ulcerative colitis.**

**Discuss the features and management of Crohn's disease.**

**Differentiate between Ulcerative colitis and Crohn's disease.**

**Ans.**

Ulcerative colitis	Crohn's disease
Both have equal incidence in males and females Both are more common in women who use OC pills Both are equally premalignant and predispose to carcinoma colon Have strong familial association	
<b>Risk factors</b>	
Smoking and appendectomy are protective	Smoking predisposes
Infections – <i>C.difficile</i> , <i>C.jejuni</i>	<i>M. paratuberculosis</i> , Measles virus
Ch. 12q – IBD-2 locus	Ch.16q- IBD1 locus
pANCA positive	ASCA positive
<b>Pathology</b>	
Continuous involvement	Skip lesions
Only large bowel involved with/out backwash ileitis	Mouth to anus with relative rectal sparing
Earliest finding is blurring of mucosal stripe and granular appearance	Earliest lesion – superficial aphthous ulcer
Mucosa and submucosa involved	Transmural involvement
Commonly rectum	Commonly ileum and ascending colon
Fibroses rare	Fibroses common
Crypt abscess, crypt branching seen	Noncaseating granulomas common
Submucosa is narrowed	Submucosa is widened

Contd...

Contd...

Ulcerative colitis	Crohn's disease
<b>Clinical features</b>	
Diarrhea is more frequent and more severe	Less common
Commonly contain mucus, pus or blood	Less common
<b>Complications</b>	
Stricture less common, +nce suggests malignancy.	Stricture more common
Toxic megacolon occurs	Rarely occurs
Fistulas are extremely rare	Very common
Malignant change can occur	Malignant change can occur.
<b>Extra-intestinal manifestations</b>	
m.c. manifestation in both is erythema nodosum. It is most responsive to treatment of IBD. Persistence of it suggests inadequate treatment	
Ulcerative colitis is more commonly associated with the extra-intestinal manifestations Pyoderma gangrenosum is more common. Primary sclerosing cholangitis is also more common with UC.	Erythema nodosum, peripheral arthritis, cholelithiasis, renal stones, ankylosing spondylitis are more common.
<b>Common radiological appearance</b>	
Garden hose appearance	Hose pipe appearance
Pseudopolyps	Cobblestone appearance
Pipestem colon	String sign of Kantor
	Raspberry/rosethorn appearance
	Halo sign on CT
<b>Surgery</b>	
Surgery is curative	Surgery is curative
Ileal pouch can be constructed	Ileal pouch is associated with many complications and therefore not constructed
Recurrence is less common	Recurrences are very common

**Medical management of IBD**

Done in two phases – Induction phase and remission phase

- 5-ASA derivatives are used in induction phase. These include sulfasalazine, balsalazine, olsalazine and mesalamine.
- Steroids can also be used for induction of remission.
- In maintenance phase, azathioprine or 6-mercaptopurine is preferred.
- Infliximab is useful mainly in fistulizing Crohn's.

**Indications of surgery**

- Intractability
- Dysplasia/carcinoma
- Toxic megacolon
- Massive GI bleeding/perforation

**Surgeries**

- Total proctocolectomy with ileal-pouch-anal anastomosis
  - Gold standard **elective procedure**
  - 2 Techniques
    - IPAA and anal mucosectomy with hand sewn anastomosis
    - IPAA with double stapled anastomosis
- Open or laparoscopic total proctocolectomy with End-ileostomy
  - *Indications*
    - Elderly
    - Incontinent patients
- Colectomy with hartmann's closure of rectum or mucous fistula
  - *Indications*
    - Acutely ill patient (fulminant colitis or toxic megacolon)
    - Pre-operative difficulty differentiating between UC and Crohn's

**Q28. Enumerate the causes of lower GI bleeding and discuss its management in brief.****Ans.****(Imp : always remember :** GI bleed terminology

- Hematemesis – vomiting of blood (upper GI bleed)
- Melena – black, tarry, sticky, foul smelling stools in patients with bleeding of atleast 60 ml blood from a site above the ligament of treitz and transit time of at least 14 hours
- Hematochezia – bright red blood in stool.
- Rectorrhagia/bleeding PR – only blood per rectum without stool.)

**Lower GI bleed**

Bleeding below the ligament of treitz

*Causes*

Colonic (95%)	Small intestinal (5%)
Diverticular disease (m.c.)	Angiodysplasia
Anorectal disease	Erosions/ulcers (enteric, TB)
Ischemic colitis, radiation colitis	Radiation enteritis
Neoplasm/colitis	Meckel's diverticulum
Inflammatory bowel disease	
Post polypectomy	

Always remember to **rule out upper GI bleed as it is the most common cause of lower GI bleed**

- m.c. cause of scanty lower GI bleed—Hemorrhoid
- m.c. cause of significant lower GI bleed—Diverticular disease
- m.c. cause of significant small bowel bleed—Angiodysplasia
- m.c. cause of scanty, recurrent, obscure lower GI bleed—Angiodysplasia (vascular ectasia).

### Management

If the patient is unstable, then proceed directly to surgery after resuscitation or perform damage control surgery as a part of resuscitation.

In such cases, if patient gives time, perform on table enteroscopy to identify the site of bleed. If still the site of bleed is not identified, proceed to serial clamping and resection or right hemicolectomy first. If still bleeding, then there is no option but to perform a total colectomy.

Direct performance of total colectomy without above mentioned steps is no longer considered rational.

For stable patients, manage according to the following steps:

#### *Step 1 - Resuscitation*

Fluid balance, electrolyte management and correction of acid-base balance once the fluid balance is taken care of are the life saving measures before surgery.

- Nasogastric tube placement for **ruling out upper GI bleeding**
- Send blood for coagulation parameters and platelets to rule out bleeding diathesis
- Monitoring of urine output, intake of fluids, pulse, blood pressure, temperature and respiratory rate is very important during this phase of resuscitation
- If necessary, a central venous line can be inserted to give fluid as per central venous pressure measurement
- Always send blood for cross matching and blood grouping to have blood available for transfusion during resuscitation or surgery
- Antibiotics are to be given if the patient is in sepsis or if he is planned for operation.

#### *Step 2 – Colonoscopy V/S Tagged RBC scan (technetium labelled RBC scan)*

- **Tagged RBC scan** is the most sensitive (can detect even 0.1 ml/min bleed) investigation to identify the presence of lower GI bleed and is to be **done whenever patient has major active bleeding but is stable**. It is of no use once bleeding has stopped. **Also it has very poor ability to spatially localize the site of bleed**
- **Option to tagged RBC scan is mesenteric angiography** which is also highly sensitive (can detect ongoing bleed of 0.5 ml/min). It has benefit of being able to carry out interventions at the same time as diagnoses such as intra- arterial vasopressin infusion or embolization of the bleeding site
- Minor bleeding or bleeding which has stopped cannot be picked up by tagged scan and in these cases, **colonoscopy** is to be performed
  - If the site is not identified but the patient stops bleeding, repeat these investigations if patient rebleeds
  - If the site is identified, then angiography and embolization or endoscopic management or surgical management can be done as per the cause of the bleed
  - If the colonoscopy and/or tagged scan are negative and the patient is still bleeding proceed to next series of investigations (**Step 3**).

#### *Step 3 – identification of bleed site in small bowel*

Small bowel series/enteroclysis/capsule endoscopy/enteroscopy are to be used to identify the site of bleed in the small bowel. These are all the necessary tests and management measures to manage a patient with lower GI bleed.

**Indications of surgery in case of gastrointestinal bleeding (Both UGI and LGI bleed)**

- Hemodynamic instability despite vigorous resuscitation (> 6 units transfusion)
- Failure of endoscopic techniques to stop bleeding
- Third recurrence after endoscopic control of bleeding
- Continued slow bleeding with transfusion requirement exceeding 3 units/day
- Shock associated with recurrent hemorrhage

**Q29. What is sigmoid volvulus? Discuss its management.****Write a note on sigmoid volvulus.**

**Ans.**

**Definition**

The condition in which the bowel becomes twisted on its mesenteric axis, a situation that results in partial or complete obstruction of the bowel lumen and a variable degree of impairment of its blood supply.

**Etiology**

- A long and floppy mesentery that is fixed to the retroperitoneum by a narrow base of origin
- Chronic constipation
- Aging (Seventh to eighth decade of life)
- Increased incidence of the condition in institutionalized patients afflicted with neuropsychiatric conditions and treated with psychotropic drugs. These medications may predispose to volvulus by affecting intestinal motility
- Diet high in fiber and vegetables
- Site - Sigmoid colon (m.c.) > The right colon and terminal ileum (*cecal volvulus*) > The cecum alone (*cecal bascule*) > transverse colon.

**Clinical features**

- Present as acute or subacute intestinal obstruction (describe characteristic features)
- Severe abdominal pain, rebound tenderness, and tachycardia are ominous signs and suggest ischemia.

**Imaging characteristics**

- Abdominal X-rays—**Bent inner tube or coffee bean appearance**
- An air-fluid level may be seen in the dilated loop of colon and gas is usually absent from the rectum
- CT—a characteristic **mesenteric whorl** is seen
- A contrast enema—**Bird's beak deformity, ace of spades or bird of prey deformity.**

**Management**

- Resuscitation
- Patients with signs of colonic necrosis should directly undergo surgery
- Nonoperative decompression (Achieved by placement of a rectal tube through a rigid proctoscope, but more often a flexible sigmoidoscope is used)
- The reduction should be confirmed with an abdominal radiograph



- The rectal tube should be taped to the thigh and left in place for 1 or 2 days to allow continued decompression and prevent immediate recurrence of the volvulus
- If detorsion of the volvulus cannot be accomplished with a rectal tube or flexible sigmoidoscope and in patients with peritonitis, laparotomy with resection of the sigmoid colon Hartmann's operation or resection with primary anastomosis, with or without protection from a proximal ostomy (transverse colostomy or ileostomy) is required
- Colonoscopy should be performed before elective resection to exclude an associated neoplasm
- Elective sigmoid resection (resection with primary anastomosis) is indicated even in patients with successful detorsion because of a prohibitive rate of recurrence after detorsion alone.

**Q30. Write a note on diverticulitis (Peridiverticulitis).**

**Ans.**

- A misnomer as it is actually a perforation of a colonic diverticulum which leads to extraluminal pericolic infection due to the extravasation of feces.
- m.c. site - Sigmoid colon

**Clinical features**

- Left lower quadrant abdominal pain that may radiate to the suprapubic area, left groin, or back
- Alterations in bowel habits
- Fever, chills, and urinary urgency
- Rectal bleeding is not usually associated
- Tenderness of the left lower abdomen, voluntary guarding of the left abdominal musculature and a tender mass in the left lower abdomen is suggestive of a phlegmon or abscess
- Abdominal wall distention if there is associated ileus or small bowel obstruction secondary to the inflammatory process
- A rectal or vaginal examination may reveal a tender fluctuant mass typical of a pelvic abscess.

**Diagnoses**

- Diagnosis based on careful history and physical examination - Begin treatment with antibiotics.

In case of doubt following diagnostic tests should be carried out:

- Computed tomography (CT) of the abdomen - reliably reveals the location of the infection, extent of the inflammatory process, presence and location of an abscess, and sympathetic involvement of other organs with secondary complications such as ureteral obstruction or a fistula to the bladder. In addition, an abscess detected by CT may often be drained by a percutaneous approach with the aid of CT guidance
- Magnetic resonance imaging (MRI)
- Water-soluble contrast enema.

**Hinchey's classification**

- Stage I: Pericolic or mesenteric abscess

- Stage II: Walled-off pelvic abscess
- Stage III: Generalized purulent peritonitis
- Stage IV: Generalized fecal peritonitis

**Management**

Uncomplicated Diverticulitis 1st episode (Disease not associated with free intraperitoneal perforation, fistula formation, or obstruction)

- Antibiotics oral or IV antibiotics
- Marked improvement in symptoms within 48 hours
- After the symptoms have subsided for at least 3 weeks, colonoscopy should be conducted to establish the presence of diverticula and to exclude cancer, which can mimic diverticulitis
- High fiber diet should be started.

Recurrent attacks of diverticulitis (>2 attacks) - surgical treatment except in immuno-compromised patients where second attack itself is an indication for surgery.

**Complicated diverticulitis**

- **Abscess**
  - Drain by a percutaneous route guided by CT or ultrasound or transrectal approach
  - IV antibiotics
  - Elective surgery approximately 6 weeks after drainage of the abscess.
- **Generalized peritonitis**
  - Hartmann's operation – resection and proximal taken out as stoma and distal closed and left inside
  - IV antibiotics
  - Appropriate generalized and nutritional support
  - Restoration of intestinal continuity to be done after minimum 10 weeks.
- **Fistula** (Diverticulitis is a more common cause of a fistula between the colon and bladder than Crohn's disease or cancer).
  - Antibiotics
  - Rule out cancer
  - A one-stage operation, taking down the fistula and excising the sigmoid colon and then fashioning an anastomosis between the descending colon and rectum.
- **Obstruction**
  - Pass a nasogastric tube to relieve the upper intestinal secretions
  - Antibiotics
  - Percutaneous drainage of the abscess.

**Q31. Write a note on Hirschsprung's disease (congenital megacolon).**

**Enumerate the causes of megacolon. Discuss Hirschsprung's disease.**

**Ans.** Megacolon is term used when the maximum diameter of colon exceeds a set value

- Cecum - >12 cm
- Ascending colon - > 8 cm
- Transverse colon - > 5.5 cm
- Rectosigmoid or descending colon - > 6.5 cm.

**Causes of megacolon**

- **Acute Megacolon** – same as pseudo-obstruction
- **Chronic megacolon** – neurologic, metabolic and systemic diseases
  - Congenital** – Hirschsprung's disease
  - Acquired nontoxic megacolon** – Chagas disease, Parkinson's disease
- **Toxic megacolon**
  - Ulcerative colitis, Crohn's colitis, Pseudomembranous colitis
  - Drugs—Narcotics (Morphine, codeine), anticholinergics (scopolamine, atropine), antipsychotics (risperidone)

**Hirschsprung's disease**

- Incidence : 1 in 5000 live births
- Boys affected four times more frequently than girls.

**Pathogenesis**

- Absence of ganglion cells in the myenteric (Auerbach) and submucosal (Meissner) plexus
- The abnormal bowel is the contracted distal segment, whereas the normal bowel is the proximal dilated portion
- Down syndrome
- Family history (chromosome 10 - RET oncogene)
- Most common site is the rectosigmoid (80%) > splenic or transverse colon > entire colon.

**Clinical presentation**

- Failure to pass meconium within the first 24 hours of life
- Progressive abdominal distention
- Bilious vomiting
- Diarrhea, fever, hematochezia and peritonitis suggest development of necrotizing enterocolitis
- Older age, poor feeding, chronic abdominal distention and significant constipation are seen.

**Diagnosis**

- **Barium enema.**
  - Failure to evacuate the instilled contrast completely after 24 hours would also be indicative of Hirschsprung's disease.
- **Manometry** – loss of rectoanal inhibitory reflex is seen.
- **Rectal biopsy**
  - Gold standard
  - Obtain the sample at least 2 cm above the dentate line.
  - Absent ganglia, hypertrophied nerve trunks, immunostaining for acetylcholinesterase (AChE), loss of calretinin immunostaining are the criteria to look for.

**Surgical management**

- Duhamel procedure - The aganglionic rectal stump is left in place and the ganglionated normal colon is pulled behind the stump

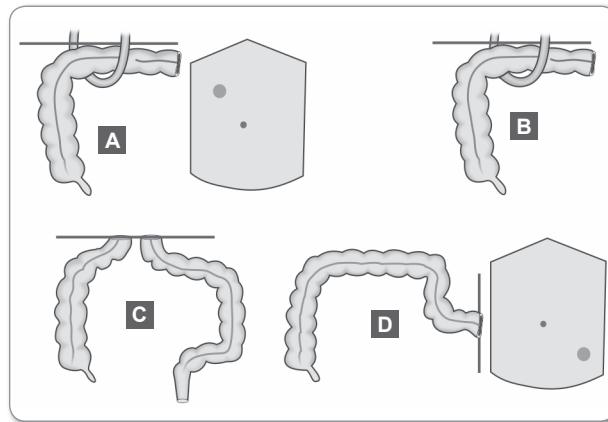
- Swenson procedure - The aganglionic bowel is removed down to the level of the internal sphincters and a coloanal anastomosis is performed
- Soave technique involves an endorectal mucosal dissection within the aganglionic distal rectum. The normally ganglionated colon is then pulled through the remnant muscular cuff and a coloanal anastomosis is performed
- Postoperative complications include constipation (m.c.), soiling, incontinence and enterocolitis.

**Q32. What are the different types of stomas that are used in surgery? Enumerate the indications for bowel stomas.**

**Ans.** Stoma can be of following types

- Urostomy—for draining of urine in case of radical cystectomy
- GI stomas—Jejunostomy, ileostomy, colostomy.

We will keep our discussion in this section on fecal diversion.



**Fig. 7:** Types of stoma

**Types 1 (Depending on the purpose for which the stoma is constructed)**

- **Diversion stoma** – done to protect distal anastomosis (Ileostomy in cases with distal ileotransverse anastomosis or low colorectal anastomosis) or to exclude an obstructed system (Distal mass)
- **Defunctioning stoma** – In cases with distal diseased bowel to give bowel rest to that part of bowel (In cases of abdominal tuberculosis).

**Types 2 (Depending on the part of bowel used)**

- Duodenostomy
- Jejunostomy – Watery clear dark green effluent start around 36 to 48 hrs after surgery
- Ileostomy – Semisolid contents, dark green effluent
- Colostomy – formed fecal matter.

**Types 3 (Depending on the duration for which it is constructed)**

- Temporary stoma (diversion stoma for colorectal or ileotransverse anastomosis)
- Permanent stoma (When entire distal bowel is resected such as abdominoperineal excision of rectum).

**Type 4 (Depending on the anatomy of the stoma constructed)**

- End stoma (Permanent stomas are usually end stoma, hartmann operation, for radiation proctitis, incontinence, perianal sepsis, initial step in the surgery of anorectal malformation)
- Loop stoma (emergency perforation surgeries wherein the site of perforation is brought out as a stoma)
- Double barrel stoma – the two separate ends are brought out together and their mesenteries united. This avoids a laparotomy during stoma closure.

**Type 5 (Depending on the continence mechanism)**

- Continent ileostomy (Kock's pouch) – has a sphincter mechanism to provide continence
- Noncontinent ileostomy (Brooke's end ileostomy, loop ileostomy etc.)

**Indications for bowel stomas***Ileostomy*

- Surgeries for enteric perforation, abdominal tuberculosis in emergency
- Ulcerative colitis, Crohn's disease, familial adenomatous polyposis
- Traumatic bowel perforation with sepsis
- Radiation enteritis, mesenteric ischemia cases
- Anastomotic leakage cases.

*Colostomy*

- Colorectal malignancy – emergency hartmann for obstruction or perforation or during elective surgery
- Fecal incontinence, volvulus surgery, rectovaginal fistulas
- Anal sphincter injury
- Complex and high anorectal malformation surgery
- Radiation proctitis, diverticulitis surgery
- Blunt or penetrating or iatrogenic trauma to colon/rectum
- Anastomotic leakage cases.

**Q. What are the complications of stomas? Outline their management in brief.**

**Ans.** According to the type of stoma

Ileostomy	Colostomy
Skin irritation (m.c.) Stoma diarrhea (2nd m.c.) Wound infection Stoma bleeding Stoma retraction Stoma prolapse	Skin irritation (m.c.) Wound infection Stoma stenosis, necrosis, retraction, prolapsed Parastomal hernia Stoma bleeding Fistula formation

**According to the duration after stoma after which it occurs**

Type	Early	Late
Stoma related	<b>Stoma necrosis</b> (within 24 hours due to impaired blood supply)	<b>Stoma stenosis</b> means narrowing of the lumen at skin or fascia level
	<b>Mucocutaneous separation</b> (Separation of skin and mucosa stitches)	<b>Stoma prolapse</b> (stoma falls externally outside of the abdomen)

Contd...

Contd...

Type	Early	Late
	<b>Stoma or peristomal skin retraction</b> (retraction means it does not protrude beyond skin and falls back towards the abdominal cavity)	<b>Parastomal hernia</b> through fascial defect adjacent the stoma loop
	<b>Stoma trauma</b> (Due to appliance, handling)	<b>Enterocutaneous fistula</b>
Peristomal skin related	<b>Excoriations</b> (acidity or alkalinity of effluent) <b>Contact dermatitis</b> (appliance or barrier material) <b>Irritant dermatitis</b> (effluent)	<b>Parastomal candidiases</b> <b>Pseudoverrucous hyperplasia</b> <b>Folliculitis</b>
Due to old disease	<b>Parastomal varices</b> in patients with portal hypertension <b>Pyoderma gangranosum</b> in patients with inflammatory bowel disease	
Metabolic and systemic effects	<b>Fluid and electrolyte imbalance</b> <b>Malnutrition</b> <b>High output stoma</b> <b>Stoma obstruction</b> <b>Gas and odor</b>	

**Management of a patient with stoma and its complications** start from the day the stoma is planned. Salient points in this regard are as follows:

- Preoperative **counselling** of the patient on the ways to manage a stoma, possible life style changes necessary after a stoma and appliance management should be done and this should be continued postoperatively
- **Stoma site** should be marked such that the stoma is visible to eye in sitting, standing and supine position. It should not fall on belt line, bony prominences, fatty prominences, scars and natural creases and should be within the rectus muscle
- Two things to use for stoma are the **stoma appliance (single piece or two piece or stoma belt)** and **Skin protective barriers**
  - Flange is a solid skin barrier which is a part of one piece or two piece appliance. This is applied at stoma site after cleaning the site with tincture iodine and allowing it to dry completely
  - Application of flange require the use of tube skin barrier paste for snug adhesion and additional skin protection
- Postoperatively, the **stoma appliance** should be applied and patient must be taught how to apply the appliance. Also the use of **skin barrier** to protect the skin should be taught to the patient. Skin barrier tubes and bottles can be powder form, paste form and liquid form for application and standard fit or precut and cut to fit solid flange when a two piece appliance is used. These skin barriers are applied around the stomas and the groove on their surface is the region on which the bag can be applied when using two piece appliance
- Patient should be advised on facts about consuming small **food** quantity frequently, avoiding water with food, avoiding foods that produce odour (onion, garlic, asparagus) and food that produce gas (beans, broccoli, cabbage, cauliflower, onions). They should also be taught to consume adequate electrolyte rich fluids to replace GI loss and also Regulate the consistency of effluent with help of foods that thicken consistency (Banana,

pasta, bread, rice, potato) and foods that liquefy the effluent (Alcoholic beverages, heavily spiced foods)

- The complications mentioned above should be managed as soon as possible to avoid longstanding problems
- Many of the **skin problems** can be managed by using barrier creams, preventing skin contact with effluent and local hygiene and a properly fitting stoma appliance
- Skin level ischemic necrosis can resolve with time and may not require surgery. Ischemic necrosis beyond the fascial level or mucocutaneous separation or stoma prolapse usually can be managed with local revision procedure and do not usually require a laparotomy
- Whereas, recurrent stoma prolapse, prolapse with parastomal hernia, fascial stenosis, complicated parastomal hernias and Peristomal fistulas usually mandate a laparotomy with correction of the problem and relocation of stoma or restoration of bowel continuity if it is possible and already planned
- Psychological support, family and peer support and vocational, emotional and social rehabilitation all play an important part in the management of a patient with stoma.

## APPENDIX

### Q33. Write the points of alvarado score and its significance.

**Ans.** Alvarado score (MANTRELS) is as follows:

- Migratory pain—1 point
- Anorexia—1 point
- Nausea/vomiting—1 point
- **Tenderness—2 point**
- Rebound tenderness—1 point
- Elevated temperature—1 point
- **Leucocytosis—2 point**
- Shift to left in leucocytosis—1 point.

**Total score – 10 points.**

9–10	Operate
7–8	High possibility – role of investigations (CT)
5–6	Possible, nice to rule out
< 5	Not possible, consider other causes/discharge

### Q34. What are the types of appendicitis? Enumerate the differential diagnoses of acute appendicitis.

**Ans. Types of appendicitis**

The appendicitis is divided into three types

- **Acute Catarrhal appendicitis:** Luminal obstruction leads to mucus retention and bacterial infection lead to increased intraluminal pressure in the appendix which leads to appendiceal wall edema followed by vascular congestion. This stage is called catarrhal appendicitis.

- **Acute phlegmonous appendicitis:** If the above condition progresses, the vascular congestion and wall edema increase more and also lead to multiple small abscess formation in the wall of the appendicitis. This is called acute phlegmonous appendicitis.
- **Acute gangrenous appendicitis:** If the above condition progresses, it leads to circulatory compromise and finally infarction at site opposite the junction of mesoappendix and appendix which is the site with the least blood supply. At this site, the appendix becomes congested red and black necrotic areas. This is called acute gangrenous appendicitis.

### Differential diagnoses

Male	Female	Children	Elderly
<ul style="list-style-type: none"> <li>• Testicular torsion</li> <li>• Ureteric stone</li> <li>• Incarcerated inguinal hernia</li> <li>• Regional enteritis</li> </ul>	<ul style="list-style-type: none"> <li>• Ectopic pregnancy</li> <li>• Ureteric stone</li> <li>• Pelvic inflammatory disease</li> <li>• Torsion/ rupture of ovarian cyst</li> <li>• Endometrioses</li> <li>• Regional enteritis</li> </ul>	<ul style="list-style-type: none"> <li>• Mesenteric adenitis</li> <li>• Incarcerated inguinal hernia</li> <li>• Meckel's diverticulum</li> <li>• Intussusception</li> <li>• Henoch Schonlein purpura</li> </ul>	<ul style="list-style-type: none"> <li>• Malignancy of colon/Small bowel</li> <li>• Diverticulitis</li> <li>• Mesenteric ischemia</li> <li>• Leaking aortic aneurysm</li> </ul>

### Clinical signs of acute appendicitis

- **Aaron sign**—Pain in epigastrium on persistent firm pressure to right iliac fossa
- **Bastede sign**—Right iliac fossa pain on rectal insufflations with air
- **Baldwin sign**—Pain on straight leg raising test in retrocecal appendicitis
- **Bassler sign**—Sharp pain on compressing appendix between abdominal wall and iliacus
- **Cope psoas test**—Extension at hip joint produce pain in right iliac fossa in patients with retrocecal appendix
- **Cope obturator test**—Internal rotation of hip cause pain in cases of inflamed pelvic appendix
- **Dunphy sign**—Pain on coughing
- **Pointing sign**—Patient points to pain in the right iliac fossa with a finger
- **Rovsing sign**—Palpation in left iliac fossa produce tenderness in right iliac fossa
- **Tenhorn sign**—Pain caused in right iliac fossa by gentle traction of the right testicle.

### Triads in acute appendicitis

- **Dieulafoy's triad** – Hyperesthesia in the sherran triangle (triangle joining umbilicus, pubic symphysis and anterior superior iliac spine), tenderness at the McBurney's point and guarding at the McBurney's point.
- **Murphy's syndrome** – Pain in the epigastrium going to right iliac fossa followed by vomiting followed fever in that sequence is seen in acute appendicitis.

(**Always Remember:** Murphy sign and sonographic murphy sign are seen in acute cholecystitis)

### Incisions for acute appendicitis

- **McBurney/McArthur/Gridiron incision** – Line perpendicular to a line joining medial 2/3 and lateral 1/3 to anterior superior iliac spine to umbilicus. It is a muscle splitting incision.
- **Rutherford Morrison muscle cutting** is lateral extension of McArthur incision.



- **Fowler – weir extension** – Medial extension of McArthur incision
- **Lanz incision (muscle splitting)** – 2 cm below umbilicus in the midclavicular line, more cosmetic
- **Rockey Davis incision** - is centered on McBurney and is transverse muscle splitting.
- **Midline vertical incision**
- **Paramedian incision**
- **Laparoscopy**

#### **Radiologic signs of acute appendicitis**

- Barium enema – Arrowhead sign, beak sign, cecal bar sign, reverse 3 sign
- USG – Ring of fire appearance or target appearance. Probe tenderness and localized ileus. It is described as noncompressible, nonperistaltic >6 mm tubular blind ending structure with gut signature sign (break in continuity of gut signature suggests perforation)
- CT – Same as barium enema and target sign.

#### **Q35. Write a note on the management of appendicular lump.**

**Write a note on the management of various appendicular pathologies.**

**What is Ochsner – Sherren Regime? Mention its components.**

**Ans.** Management of a patient of appendicular lump is as follows:

Simple catarrhal appendicitis	Laparoscopic/open Appendicectomy	
Complicated appendicitis/ appendicular lump	CECT in adults and USG in children and pregnant women confirm lump	Ochsner – Sherren Regime
	CECT in adults and USG in children and pregnant women confirm phlegmonous appendicitis	Antibiotics for small and antibiotics with percutaneous drainage for large abscess followed by interval appendicectomy after colonoscopy in adults
	Persistent phlegmonous/ Gangrenous appendicitis	Open appendicectomy
Recovering appendicitis in adults	Interval appendicectomy after colonoscopy	
Recovering appendicitis in children	Interval appendicectomy	

#### **Components of Ochsner-Sherren regime include the following:**

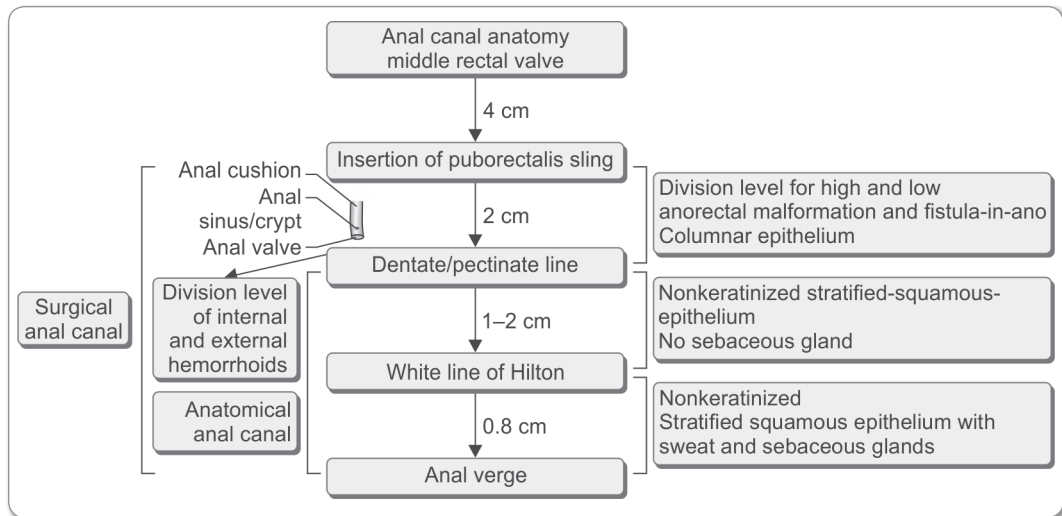
- Nil per oral
- IV fluids and antibiotics
- Analgesia and antacids
- Pain scale evaluation and charting
- Monitoring of pulse, blood pressure, temperature, size of mass and intake – output of the patient.

## RECTUM AND ANAL CANAL

**Q36. Describe the anatomy of anal canal and significance of dentate line.**

**Draw a diagram showing the anatomy of the anal canal.**

**Ans.** The figure below shows all the important points to remember in relation to anatomy of anal canal.



**Fig. 8: Anal canal anatomy**

- **Surgical anal canal** - All the region distal to the insertion of the levator ani muscles is the anal canal. The surgical anal canal includes the anatomic anal canal and the distal 2 cm of the rectum above the pectinate line. Total length is 4 cm.
- **Anatomical anal canal** – Part distal to pectinate line (dentate line). Total length is 2 cm.

### Changes in dentate line according to dentate line (pectinate line)

	Below the dentate/pectinate Line	Above the dentate/pectinate Line
Embryonic origin	Ectoderm	Endoderm
Epithelial lining	Stratified squamous epithelium	Simple columnar epithelium
Arterial supply	Inferior rectal artery	Superior rectal artery
Venous drainage	Systemic, by way of inferior rectal vein	Portal, by way of superior rectal vein
Lymphatic drainage	To inguinal nodes	To pelvic and lumbar nodes
Nerve supply	Inferior rectal nerves (somatic)	Autonomic fibers (visceral)

### Anorectal ring

The puborectalis with the superficial and deep parts of the external sphincter and the proximal part of the internal sphincter form the so-called anorectal ring. This ring can be

palpated and since cutting through it will produce incontinence, it must be identified and protected during surgical procedures.

The **anal verge** is the junction of skin around the anal opening and the anal mucosa.

The **dentate or pectinate line** is located 2 cm above the anal verge and represents the junction between the anal transition zone below and the anal nonkeratinizing squamous mucosa.

**Q. Discuss the anatomy of anal sphincters and explain their role in continence mechanism.**

**Ans. Anal Sphincters**

Internal anal sphincter	External anal sphincter
Circular (inner) muscle coat	Longitudinal (outer) muscle layer with externally, the fibers of levator ani.
The lower end of internal sphincter lies lower than the external sphincter	Uppermost is puborectalis sling
Muscle that is cut during sphincterotomy and exposed during hemorrhoidectomy	Important part played by it in continence mechanism

**Continence mechanisms**

- Anal canal lengthens with squeezing of the external sphincter and shortens with straining
- **Resting pressure** of anal canal is due to the tone of internal anal sphincter and is approximately 90 cm water or 40 mm Hg
- **Squeeze pressure** is 40 to 80 mm Hg greater than resting pressure and is produced by contraction of the external sphincter the maximum time for which is one minute
- **High pressure zone** is 2 to 4 cm
- The principle mechanism that provides continence is pressure differential between rectum (6 cm water) and anal canal (90 cm water)
- **Anorectal angle** ( $N-110^\circ$ ) acts like a flap valve mechanism
- Pelvic floor sensory receptors differentiate anorectal sensation of gas, liquids and solids. This sensation persists after proctectomy and ileoanal anastomosis
- **Rectoanal inhibitory reflex (RAIR)** – Increase in anal tone followed by brief relaxation of internal sphincter transiently after proximal rectal dissection to sense the contents is called rectoanal inhibitory reflex.

The rectoanal inhibitory reflex is absent in Hirschsprung's disease and after coloanal anastomosis.

- Other important factors in maintenance of continence are:
  - Rectal capacity
  - Compliance and tone of rectum and anal canal
  - Stool volume and consistency.

**Q37. Draw a diagram showing the blood supply of the rectum.**

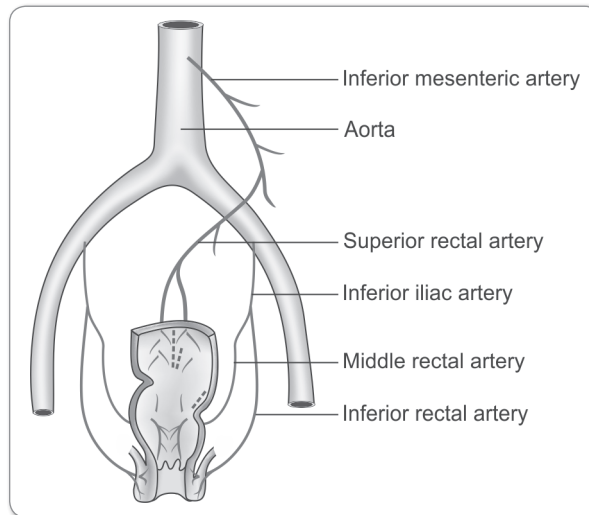
**Discuss blood supply of rectum.**

**Ans. Arterial supply**

The arteries of the rectum and anal canal are the unpaired superior rectal artery, the paired middle and inferior rectal arteries and the median sacral arteries.

- The **superior rectal (hemorrhoidal) artery** arises from the inferior mesenteric artery and descends to the posterior wall of the upper rectum. Supplying the posterior wall, it divides and sends right and left branches to the lateral walls of the middle portion of the rectum down to the pectinate (dentate) line
- The **middle rectal (hemorrhoidal) arteries** are always present in the lateral rectal stalks and originate directly or often indirectly from the anterior division of the internal iliac artery
- The **inferior rectal (hemorrhoidal) arteries** arise from the internal pudendal branch of the anterior division of the internal iliac arteries and proceed ventrally and medially to supply the anal canal distal to the pectinate line
- The **median sacral artery** arises just above the bifurcation of the aorta and descends beneath the peritoneum on the anterior surface of the lower lumbar vertebrae, the sacrum and the coccyx. It sends several very small branches to the posterior wall of the rectum.

**The venous drainage follows arterial supply so that** the middle and inferior rectal veins drain into the internal iliac system and the superior rectal vein drains into the inferior mesenteric vein which opens into the portal circulation via the splenic vein.



**Fig. 9:** Blood supply of rectum

**Q38. Write a note on Fissure-in-ano.**

**Discuss management of a patient with Fissure-in-ano.**

**Ans. Introduction**

- An anal fissure is a linear ulcer of the lower half of the anal canal, usually located in the posterior commissure in the midline.

**Etiological factors**

- Passage of large hard stools
- Previous anal surgery

- Reduced anal blood flow in the posterior midline
- Childbirth
- Laxative abuse
- Higher than normal resting anal canal pressures.

**Clinical features**

- Perianal pain which increases with defecation and straining
- Bleeding per rectum
- Prior history of constipation
- Most fissures in women and men are in the posterior midline
- An anterior midline fissure is seen more often in women
- **Chronic fissure (> 4 to 6 weeks duration)**
  - A sentinel pile or tag externally and an enlarged anal papilla internally.
- **Atypical fissure (other than these locations and nonhealing fissures)**

Rule out Crohn's disease, tuberculosis, malignancy, hidradenitis suppurativa or an STD.

**Diagnoses**

- These lesions actually involve just the anal tissues and are typically best seen by visually inspecting the anal verge with gentle separation of the gluteal cleft
- Digital rectal exam and proctoscopy are extremely painful and not to be done.

**Management***Nonoperative management*

- High fiber diet
- Mild laxatives and fiber supplements
- Warm and moist perianal sitz baths
- Topical nitric oxide donors (e.g. nitroglycerin)
- Calcium channel blockers (e.g. diltiazem, nifedipine)

For nitroglycerin, the limiting side effects are headaches and tachyphylaxis. The topical application of diltiazem (2%) produces fewer side effects and similar efficacy as nitroglycerin. Fissure healing can be anticipated in approximately 70% of patients with chronic fissures using nitroglycerin or diltiazem.

- **Reversible chemical sphincterotomy** - Internal sphincter injection of botulinum toxin (Botox), a technique that transiently produces striated muscle denervation, leading to muscle paralysis and relaxation. It has been recommended as a nonsurgical treatment of fissures with a low risk for complications.

*Surgical Management***Indications**

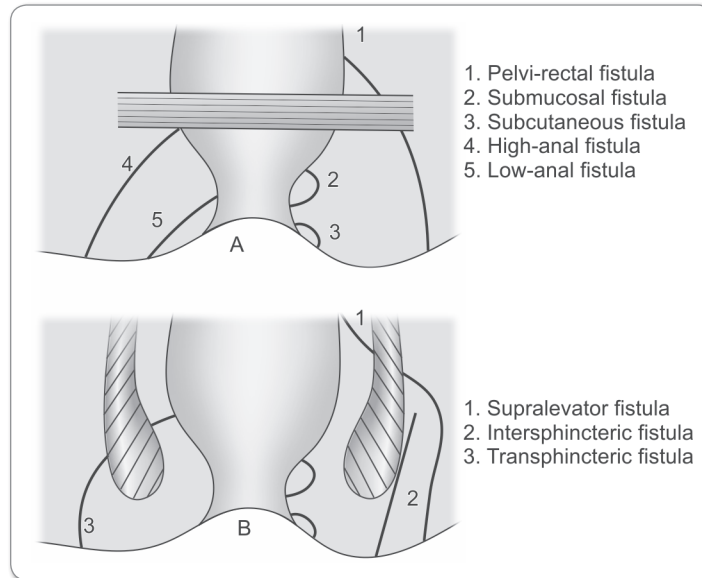
- Severe and chronic fissures
- Excruciating pain in acute fissure
- Failed medical therapy can benefit from surgery

**Procedure**

- Partial lateral internal sphincterotomy using closed or open technique (Notara's)
- Anorectal advancement flap

**Q39. Classify fistula in ano and discuss its management.**

**Ans.** Most fistulas are caused by sepsis originating in the anal canal glands at the dentate line.



**Fig. 10:** Fistula classification

**Park's classification**

- Intersphincteric (the most common): The fistula track is confined to the intersphincteric plane
- Trans-sphincteric: The fistula connects the intersphincteric plane with the ischiorectal fossa by perforating the external sphincter
- Suprasphincteric: Similar to trans-sphincteric, but the track loops over the external sphincter and perforates the levator ani
- Extrasphincteric: The track passes from the rectum to perineal skin, completely external to the sphincteric complex.

Other classification is **St. James University hospital MR imaging classification**

Stage	St. James University hospital MR imaging classification
0	Normal anatomy—no fistula
1	Intersphincteric—simple
2	Intersphincteric with secondary branching or intersphincteric abscess
3	Transsphincteric—simple
4	Transsphincteric—with secondary branching or ischiorectal or ischioanal fossa abscess
5	Suprasphincteric/extrasphincteric/translevator fistula

**Management**

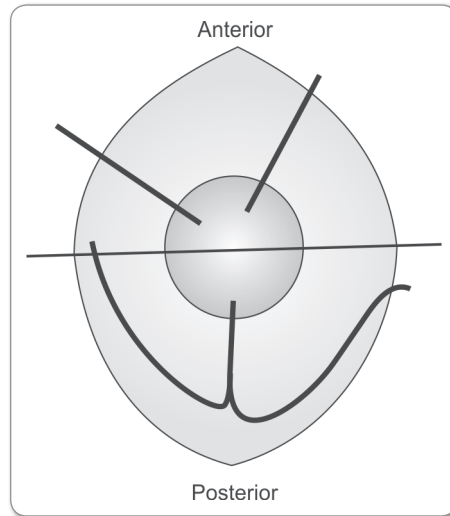
*On digital examination*

Subcutaneous induration may be traced from the external opening to the anal canal.

- A palpable nodule in the wall of the anal canal

- A probe can be eased gently, not forcefully, from the external skin opening to the internal anal canal opening.

**Goodsall rule** applied to identify the site of internal opening as follows:



**Fig. 11:** Goodsall rule

- The rule predicts that if a line is drawn transversely across the anus, an external opening anterior to this line will lead to a straight radial tract, whereas an external opening that lies posterior to the line will lead to a curved tract and an internal opening in the posterior commissure
- The long anterior fistula is an exception to the rule
- Also when both the anterior and posterior openings are present the rule of the posterior opening applies.

**MR fistulogram** is the investigation of choice.

(**IMP** : In case of discharging fistulas MR fistulogram is a T2 image without any contrast which will give the results for identification of the tract and secondary branching. In case of a non discharging chronic fistula, saline injection and T2 images is called MR fistulogram.)

### Treatment

#### Principles of surgery

- Identification of the fistula tract, both the openings and secondary tracts
- Excision of the entire tract
- Preservation of the sphincter function

#### Options

- For superficial fistulas involving small quantities of sphincter muscle, primary fistulotomy is simple and definitive
- For anterior fistulas in women and fistulas involving more than 25 to 50% of the bulk of sphincter muscles, **Seton** placement (silk or nylon suture) should be preferred over primary fistulotomy
- Sitz baths and wound dressing

For complex, deep or recurrent fistulas, the newer techniques are as follows:

- Video-assisted anal fistula treatment (**VAAFT**) – a novel minimally invasive and sphincter-saving technique for treating complex fistulas. Here, using an endoscope, the fistula tract is directly visualized and destruction of the fistula tract is done from the inside followed by cleansing of the fistula tract and finally closure of the internal opening
- Ligation of the intersphincteric fistula tract (**the LIFT procedure**) - This approach consists of ligation of the tract in the intersphincteric space, curettage of the tract and closure of the external anal sphincter defect with sutures
- Fistula laser closure (**FILaC**) - This combines conventional closure of the internal opening using a flap with laser obliteration of the fistula tract
- Anal fistula plugs and fibrin glues.

The short hospital stay (same day discharge), the short recovery time following the operation and the short absence from work result in relative cost effectiveness of the VAAFT procedure and make it a preferred technique in the patients with complex fistula.

**Q40. Write a note on Pilonidal Disease (Jeep driver's disease, Barber's disease).**

**Ans.**

**Introduction**

- Latin : pilus – hair and nidus – nest
- It is basically a subcutaneous tract containing hairs and other debris
- Typically occur in the midline of the sacrococcygeal skin of young men
- It has noninfected, midline openings communicating through a fibrous tract and lined by granulation tissue and stratified squamous epithelium with free hair lying loosely within the lumen or within the granulation tissue.

**Etiopathogenesis**

- Hair seems to play a central role in the process of infection and perpetuation of granulation tissue in sinuses.
- Pilonidal patients are often dark haired hairy males.
- Buttock friction and shearing forces in the natal cleft cause the shed hair to be entrapped in the natal cleft by suction forces and create a subcutaneous chronically infected midline tract.
- From this tract, over time, other secondary tracts arise and spread laterally and form branching side channels.
- Thompson's operation for lymphedema is also a predisposing factor.

**Clinical features**

- Common sites – Natal cleft strictly in midline, umbilicus, web spaces of hand, site of Thompson's operation for lymphedema
- Present with swelling and serosanguineous/purulent/blood stained discharge at the site of sinus which may have a single or multiple openings
- Intermittent pain and history of the swelling bursting spontaneously
- Tufts of hair may be seen projecting from the sinus.

**Treatment**

- Abscess to be drained down to the subcutaneous tissues off the midline.



- Hair should be removed from the wound.
- Local skin should be shaved weekly to prevent the reintroduction of hair.

**Surgical management** is done for patients who have recurring infections.

- Midline excision and primary suture. For primary closure, the omission of wound drainage is associated with a higher frequency of minor and major wound infections and wound dehiscence
- Marsupialization
  - The areas of midline pits and sinuses are removed and the wound reduced in size by suturing the wound edges to the fibrous base of the wound
  - This can reduce wound healing times and may be effective at removing extensive sinus tracts
  - Low rate of reinfection and wound breakdown
- Rhomboid excision
- Karydakis procedure
- Limberg flaps
- Oblique excisions with bilateral gluteus maximus fascia advancement flaps
- Bascom's cleft closure (Incision lateral to midline, excision of midline sinus, closure of midline wound and secondary healing for the lateral wound)
- V-Y advancement flaps can all be used for repair
- The complex flap closures are reserved for patients with refractory disease for whom simple measures have failed and are always done with complete excision of the pilonidal sinus.

### **Complications**

- Recurrence – may be due to re-entrapment of hair or inadequate excision
- Abscess formation

### **Q41. Write a note on rectal prolapse (Procidentia).**

**Discuss in brief the management of a patient with rectal prolapse.**

**Ans.**

#### **Etiology (Theories)**

- Sliding herniation of the pouch of Douglas through the pelvic floor fascia into the anterior aspect of the rectum
- Full-thickness rectal intussusception starting approximately 3 inches above the dentate line and extending beyond the anal verge
- Abnormal anatomic features—Diastasis of the levator ani, abnormally deep cul-de-sac, redundant sigmoid colon, patulous anal sphincter, and loss of the rectal sacral attachments
- Chronic or lifelong constipation with a component of straining.

#### **Clinical features**

- Women, 50 years and older are six times more common than men
- Young male patients have psychiatric disorders and many are institutionalized

- Fecal incontinence
- Prolapse initially comes down with defecation or straining and reduce spontaneously afterward
- A sensation of chronic moisture and mucous drainage in the perineal area
- Chronic cases show ulceration and significant bleeding
- 20–30% female patients also have vaginal vault prolapse and urinary incontinence might be present.

### Diagnoses

- The prolapsed tissue folds are always concentric in rectal prolapse whereas hemorrhoidal tissue develops radial invaginations defining the hemorrhoidal cushions
- Colonoscopy to rule out neoplasia as lead point
- Inspection of the perineum with the patient in the sitting or squatting position is helpful for this purpose
- Defecography
- Anal manometry and pudendal nerve terminal motor latency tests.

### Mangement

- Approaches - abdominal and perineal
- The perineal approach results in less perioperative morbidity and pain and a reduced length of hospital stay but has a higher recurrence rate. The converse is true for the abdominal approach.

### Contraindication to surgery

- Prolapse for longer than 2 years, should be warned of the possibility that incontinence could persist
- As a sole procedure to restore continence.

### Abdominal procedures

- **Ripstein** mesh anterior repair (rectopexy)
- **Wells ivalon's** procedure (posterior rectopexy)
- **Orr-Loygue** rectopexy – lateral mesh placement.
- Resection Rectopexy (**Frykman and Goldberg**).

### Perineal procedures

- **Altemeier** procedure (first done by Mickulicz)
- **Delorme** procedure - mucosectomy again through a perineal approach
- **Thiersch** anal encirclement.

## Q42. Write a note on Hemorrhoids.

### Classify hemorrhoids and discuss its management.

#### Ans. Normal anal cushions

- These are specialized, highly vascularized cushions forming discrete masses of thick submucosa containing blood vessels, smooth muscle and elastic and connective tissue.

- Common location—Left lateral, right anterior and right posterior quadrants of the canal
- Role—Aid in anal continence.

**Hemorrhoids:** Clinical situations in which these cushions are abnormal and cause symptoms.

**Types (Based on location above or below the dentate line)**

- **External hemorrhoids:** Covered with anoderm and are distal to the dentate line, painful if thrombosed
- **Internal hemorrhoids:** Painless, bright red bleeding or prolapse associated with defecation.

**Clinical features**

- Mass protruding per rectum
- Dripping of blood in the toilet bowl
- Mucus and fecal leakage and perianal pruritus
- Pain suggest external hemorrhoidal thrombosis.

**Investigations**

- Digital examination
  - Assessment of internal and external hemorrhoidal disease
  - Anal canal tone
  - Exclusion of other lesions, especially low rectal or anal canal neoplasms.
- **Investigation of choice** - Anoscopy

Grade	Appearance	Management
1st	Painless bleeding, no prolapsed	Medical management/rubber band ligation/Sclerotherapy (5% phenol in arachis or almond oil)/Infrared photocoagulation.
2nd	Prolapsed but reduce spontaneously	
3rd	Prolapsed and require manual reduction	
4th	Cannot be manually reduced	Surgery
Internoexternal hemorrhoids	Both internal and external hemorrhoids	Surgery

**Indications of colonoscopy or double contrast barium enema**

- The history is uncharacteristic
- The patient is older than 40 years
- The patient has risk factors for colon cancer, such as a positive family history.

**Treatment**

*Nonoperative Management*

- Avoidance of excessive straining
- Better dietary habits
- Fiber supplements
- Stool softeners.

*Out patient procedures*

- **Indications** – Second and some third degree internal hemorrhoids
- **Symptomatic external haemorrhoids are a contraindication to these procedures**

*Procedures*

- Rubber band ligation—Simplest and most effective. One band at a time. Care to rule out immunodeficiency/blood thinners/patients at risk of infective endocarditis
- Sclerotherapy
- Infrared coagulation
- Heater probe
- Bipolar electrocoagulation.

*Surgical Treatment***• Indications**

- Fail to respond satisfactorily to repeated attempts at conservative measures
- Grade 4 hemorrhoids
- Hemorrhoids complicated by strangulation or associated pathology, such as ulceration, fissure or fistula or hemorrhoids are associated with symptomatic external hemorrhoids or large anal tags

- Options.

**Closed hemorrhoidectomy (Ferguson)**

- Simultaneous excision of internal and external hemorrhoids

**Milligan Morgan open hemorrhoidectomy****Whitefield submucosal hemorrhoidectomy****Minimally invasive procedures for hemorrhoids (MIPH)**

- Application of ultrasonic or controlled electrical energy (harmonic scalpel, LigaSure) which remove the excess hemorrhoidal tissue and coagulate or seal the blood vessels simultaneously with minimal lateral thermal injury to nearby tissue.

**Longo's procedure (Stapled hemorrhoidectomy or stapled hemorrhoidopexy)**

- Used to treat circumferential prolapsed and bleeding haemorrhoids
- Excises a circumferential portion of the lower rectal and upper anal canal mucosa and submucosa and performs a reanastomosis with a circular stapling device.

**Q43. Discuss anorectal malformations.****Write a note on management of imperforate anus.**

**Ans.** Includes agenesis and atresia of the rectum and anus called imperforate anus.

**Embryology**

- Between 4 and 6 weeks, urorectal septum and lateral cloacal folds divide the cloaca into anterior urogenital sinus and posterior cloaca. Any defect in this division can lead to anorectal malformations.

**Etiology:** Unknown

**Incidence:** 1 in 4,500 newborns

It is more common in males.

Types of ARM in males	Types of ARM in females
<ul style="list-style-type: none"> <li>• Cutaneous (perineal fistula) (<b>bucket handle malformations/anal membrane/covered anus</b>)</li> <li>• Rectourethral fistula               <ul style="list-style-type: none"> <li>– Bulbar (<b>m.c.</b>)</li> <li>– Prostatic</li> <li>– Bladder neck fistula</li> </ul> </li> <li>• Imperforate anus without fistula</li> <li>• Rectal atresia</li> </ul>	<ul style="list-style-type: none"> <li>• Cutaneous (perineal fistula)</li> <li>• Vestibular fistula (<b>m.c.</b>)</li> <li>• Imperforate anus without fistula</li> <li>• Rectal atresia</li> <li>• Cloaca               <ul style="list-style-type: none"> <li>– Short common channel</li> <li>– Long common channel</li> </ul> </li> <li>• Complex malformations</li> </ul>

**Other classification is into Low** (rectum reaches below levator ani) suggested by perineal fistula/anal stenosis/anal membrane or midline raphe fistula and high (rectum ends above the levator ani) suggested by flat bottom and/or meconuria.

### Associated defects

- **Sacrum and Spine**
  - Sacral deformities appear to be the most frequently associated defect.
- **Genitourinary Defects**
  - The frequency of associated genitourinary defects varies from 20% to 54% and increases as the height of ARM increases.
- **VACTERL** anomalies
  - **V** Vertebral body segmentation defect
  - **A** Anal atresia
  - **C** Cardiovascular (PDA, VSD)
  - **TE** Tracheo esophageal fistula
  - **R** unilateral Renal agenesis
  - **L** Limb anomaly (radial ray hypoplasia)

### Diagnosis

- **Perineal inspection** is the most important examination and more important in females.
  - Perineal/vestibular fistula—Low type
  - Single perineal opening in females—Perineal fistula
  - Urethral and vaginal opening only—High imperforate anus
  - Anal + one other opening—Persistent urogenital sinus
- **Wangensteen Rice Invertogram**
  - Lateral pelvic radiograph taken after the infant held upside down for several minutes

### Other investigations

- Echocardiogram
- Renal ultrasound and urine analysis
- Spine radiographs
- Voiding cystourethrogram

**Mangement**

Type of ARM	Management
• Low anal stenosis	• Serial daily dilatation
• Low and anteriorly displaced anus	• Cutback anoplasty or transposition anoplasty as 1 stage procedure
• Low perineal fistula without other abnormalities and with <b>rectal gas below coccyx in invertogram</b>	• DeVries and penas posterior sagittal anorectoplasty (PSARP) with or without colostomy
• Imperforate anus with <b>rectal gas above coccyx/associated abnormalities/flat bottom/sacral abnormalities</b>	• End sigmoid colostomy with mucus fistula f/b division of fistula and penas PSARP after 3 to 6 months f/b closure of colostomy after several weeks f/b anal dilatation after 2 weeks and continued for months
• Single perineal orifice in females	• Colostomy/urinary diversion and drainage of hydrocolpos f/b management of colostomy as above.
• Complex malformations	• Individualized management

**Complications**

- Repair of low ARM – Constipation
- Repair of high ARM – Incontinence.

**Q44. Write a note on Ischiorectal abscess.**

**Describe the anatomy of ischiorectal space and discuss ischiorectal abscess.**

**Ans. Anatomy of ischiorectal fossa**

- A pyramidal space on either side of the anal canal and lower rectum
- It is posterior to the base of the urogenital diaphragm
- **Base** - Perianal skin
- **Medial wall** - External anal sphincter and levator ani
- **Lateral wall** - Internal obturator fascia
- **Apex** – Point where the levator muscles join the obturator internus muscle
- The spaces on two sides communicate posteriorly through the retrosphincteric space. Thus, **it has a horse shoe extension** to the opposite side which can allow the spread of abscess from one space to the opposite space.
- The lower portion of the ischioanal (ischiorectal) space has been termed the perianal space. Laterally it communicates with the gluteal fat and the subcutaneous space.

**There are four types of anorectal abscess** – perianal (m.c. – 60%), ischiorectal, intersphincteric and supralelevator.

**Ischiorectal abscess**

It is more common in males.

**Incidence** : 30% of all anorectal abscess

**Etiology**

**Park's cryptoglandular theory of intersphincteric anal gland infection**

Infection of anal gland is followed by formation of pus which travels along the path of least resistance.

- Caudally—perianal abscess
- Laterally across the external sphincter—Ischiorectal abscess
- Superiorly above the anorectal junction—Supralevator intermuscular or pararectal abscess (depending on its relation to the longitudinal muscle)
- Circumferentially: Intersphincteric/intermuscular, ischiorectal or pararectal supralevator abscess.

*Other sources of infection (non anal gland infections)*

- Foreign body, trauma, deep skin-related infection
- Underlying rectal disease such as neoplasm, Crohn's disease and tuberculosis
- Generalized disorders, such as diabetes and acquired immunodeficiency syndrome (AIDS)

### **Clinical features**

- Painful erythematous swelling in the region of abscess suggests perianal abscess
- Fever, and more of constitutional symptoms but less local symptoms point more towards ischiorectal abscess
- On examination, the affected buttock might be diffusely swollen and tender.

### **Investigations**

- Digital rectal exam
- Examination under anaesthesia
- Proctoscopy, sigmoidoscopy.

### **Treatment**

- Cruciate incision over the most fluctuant point with excision of the skin edges to de-roof the abscess
- Pus is sent for microbiological culture
- Tissue from the wall is sent for histological study
- With a finger in the anorectum, the cavity is carefully curetted and fistula looked for
- However, immediate fistulotomy should not be performed
- After irrigation of the cavity, the wound is lightly packed
- Antibiotics are prescribed if there is surrounding cellulitis and in those less resistant to infection, such as diabetics
- If gut flora are cultured, it is likely, but not inevitable that there is an underlying fistula.

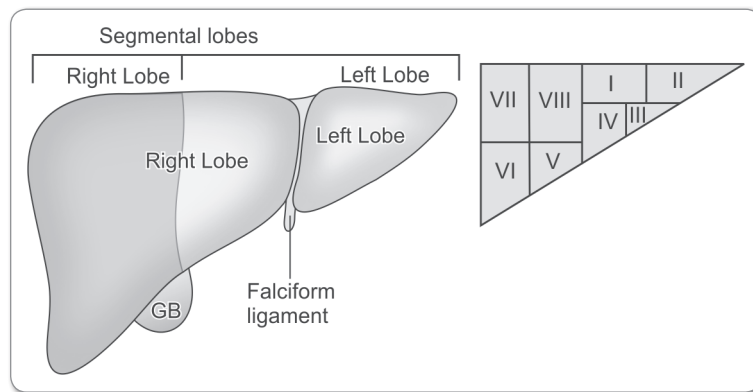
## **LIVER**

**Q45. Explain the anatomy of liver.**

**Ans.**

- Liver is the largest solid organ of the body.

- It has **three surfaces** – Anterosuperior, posterior and inferior and **four margins** – Right lateral, superior, inferior and anterior which forms the three surfaces
- **Peritoneal attachments**
  - Liver is invested in glisson capsule. This capsule also covers the structures of the portal triad at the liver hilum
  - The peritoneal investments of liver are called ligaments and can be shown in the figure as follows
- **Division of liver** anatomically forms “Lobes” and functional division gives rise to “Hemilivers”
  - Anatomical division is into right and left lobes by **Cantlie’s line** which is as shown in figure and which runs from gallbladder fossa to left side of IVC
  - Functional division (Couinaud) is along the portal scissura which run along the hepatic veins. Thus, the hepatic veins are intersegmental whereas the portal veins are intrasegmental except pars umbilicus part of left portal vein which is intersegmental. Each couinaud segments have their own portal triad.
  - **Basic** – Hemiliver—sector—segment



**Fig. 12:** Liver anatomy

There are **3 portal scissura in Couinaud nomenclature** along the 3 hepatic veins as follows:

- Right portal scissura is from right margin of IVC to inferior margin of liver midway between gallbladder fossa and right margin of liver. It contains the right hepatic vein and separated segments V and VIII from segments VI and VII
- Main portal scissura is from left side of IVC to midway between dorsal 1/3 and ventral 2/3 of left margin of liver to commencement of ligamentum venosum. It contains the middle hepatic vein and divides the liver into right and left hemilivers
- Left portal scissura runs midway between left margin and main portal scissura contains the left hepatic vein and divides the left hemiliver into segments II posteriorly and segments III and IV anteriorly.

**Functional division is also given by Strassberg which is based on bile duct and hepatic artery and not on portal vein.**

- The only difference is that strassberg divides the left lobe into medial (IV) and lateral (II, III) segments and not like in Couinaud nomenclature



- Thus right hemiliver has anterior (V,VIII) and posterior sectors (VI,VII) and left hemiliver has medial (IV) and lateral (II,III) sectors.

**Thus the segments and there names are as follows:**

1. Caudate lobe (bounded by IVC posteriorly and portal vein anteriorly with the middle and left hepatic veins superiorly as they enter into IVC and left portal vein inferiorly)
  2. Left superior lateral
  3. Left lateral inferior
  4. Left medial (quadrate lobe) A- superior, B- inferior – which is divided at the level of portal vein
  5. Right anterior inferior
  6. Right posterior inferior
  7. Right posterior superior
  8. Right anterior superior
- **Riedel lobe** is tongue like anteroinferior projection from segment V
  - **Spigelian lobe** is a part of caudate lobe anterior to ligamentum venosum called as spigelian lobe. Caudate lobe and specifically spigelian lobe hypertrophy is seen in Budd Chiari syndrome.

**Common hepatic artery**

- Arises from celiac trunk and runs along the upper border of head of pancreas
- It gives gastroduodenal artery posterosuperior to duodenum and continues in the hepatoduodenal ligament to the left of CBD and anterior to portal vein
- The relations of portal triad in the hepatoduodenal ligament in a sagittal section USG form a **mickey mouse configuration** with hepatic artery forming the left ear, CBD the Right ear and portal vein the face
- It divides into right and left hepatic arteries
- Right A gives anterior and posterior branches which each divide into superior and inferior branches to supply segments V to VIII cystic artery is a branch of right hepatic artery
- Left A gives medial and lateral branches which each divide into superior and inferior branches. Left hepatic artery sometimes gives a middle hepatic artery to segment IV.

**Hepatic veins (Central lobar vein – segmental veins – sectoral veins – hepatic veins)**

- They are not accompanied by portal triad and are intersegmental
- They have no glissonian coverings and are in direct contact with the hepatic parenchyma
- Segments V to VII drain to the right hepatic vein. The superior tributary is the largest and mainly drains segment VII
- Segments IV and V drain into the middle hepatic veins. Segment VIII also drains into the middle hepatic vein via a tributary separate from segment IV and V tributary
- Umbilical vein and segment II,III and IV drain into the left hepatic vein.

**Common bile duct**

- Right branch is shorter (1 cm) than the left (2.5 cm). It has divisions similar to the hepatic artery.

**Q46. Classify the cystic lesions of liver. Discuss the management of a patient with liver hydatid cyst.**

**Enumerate the liver cysts and discuss hydatid liver disease in brief.**

**Ans. Differential diagnoses of liver cysts**

Simple cyst		<ul style="list-style-type: none"> <li>• Benign developmental hepatic cyst</li> <li>• Caroli disease</li> <li>• Von Meyenburg complex</li> <li>• Adult polycystic liver disease</li> </ul>
Complex cyst	Neoplasm	<ul style="list-style-type: none"> <li>• Hepatocellular carcinoma</li> <li>• Biliary cystadenoma and cystadenocarcinoma</li> <li>• Cavernous hemangioma</li> <li>• Embryonal sarcoma</li> </ul>
	Inflammatory/ infectious	<ul style="list-style-type: none"> <li>• Amebic abscess</li> <li>• Pyogenic abscess</li> <li>• Hydatid cyst</li> </ul>
	Post-traumatic and miscellaneous	<ul style="list-style-type: none"> <li>• Pseudocyst</li> <li>• Hematoma</li> <li>• Biloma</li> <li>• Infected or hemorrhagic cyst</li> </ul>

### Management of hydatid liver

#### *Clinical presentation*

- The most common presenting symptoms are abdominal pain, dyspepsia and vomiting
- The most frequent sign is hepatomegaly
- Jaundice and fever.

#### *Complications*

- Bacterial superinfection
- Rupture of the cyst into the biliary tree (M.C. complication. ERCP is used to diagnose), bronchial tree or free rupture into the peritoneal, pleural or pericardial cavities can occur
- Free ruptures can result in disseminated echinococcosis and/or a potentially fatal anaphylactic reaction.

#### *Diagnoses*

- Eosinophilia
- Ultrasound
- CT or MRI scans
- In patients with suspected biliary involvement, ERCP or percutaneous transhepatic cholangiography (PTC) may be necessary.

WHO	Gharbi	Features	Type of cyst
CL		Unilocular well circumscribed with a well defined wall	Active fertile cyst
CE1	I	Unilocular cyst with double line sign (concentric hyperechogenicity) with mobile debris (hydatid sand)	
CE2	II	Multiseptated honeycomb/spoke wheel/rosette appearance	

*Contd...*

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WHO	Gharbi	Features	Type of cyst
CE3	III	Floating undulating hyperechoic membranes called water lily sign	Transitional cyst
CE4	IV	Heterogeneous contents. No daughter cysts	Inactive degenerated cyst
CE5	V	Solid and calcified amorphous mass.	Inactive dead cyst

**Treatment**

- **Primarily surgical.**

**Conservative management** - Only in older patients with small, asymptomatic, densely calcified cysts

- Treatment of echinococcosis with albendazole or mebendazole is effective at shrinking cysts in many patients with E. Granulosus.

**PAIR (puncture, aspiration, injection, and reaspiration)**

- Formalin, hypertonic saline, cetrimide, chlorhexidine, hydrogen peroxide and ethyl alcohol are some of the compounds used as scolicidal. All are concentration dependent and their degree of dilution in the cyst contents is quite unpredictable
- **Contraindications of PAIR** include inaccessible cysts in liver, very superficial cysts in liver, lung cysts, brain cysts, honeycombing, dead or inactive cysts.

**Surgery**

- Is the primary treatment for hydatid cyst. **Preoperative medical treatment** may decrease the risk of spillage and is a reasonable and safe practice
- The anesthesiologist should have epinephrine and steroids available in case of an anaphylactic reaction
- Cyst is exposed after adequate liver mobilization
- Packing off the abdomen with sponges soaked in scolicidal solution is important because rupture can result in anaphylaxis and diffuse seeding
- Usually, the cyst is then aspirated through a closed suction system and flushed with a scolicidal agent such as hypertonic saline or others mentioned above
- The cyst is then unroofed, which is followed by one of excision (or pericystectomy), marsupialization procedures, leaving the cyst open, drainage of the cyst, omentoplasty, or partial hepatectomy to encompass the cyst
- Total pericystectomy or formal partial hepatectomy can also be performed without entering the cyst
- Laparoscopic techniques for drainage and unroofing of cysts have been successful.

**Q47. Write the differential diagnoses of space occupying lesions of the liver. Mention the management of a case of amebic liver abscess.**

**(IMP: REMEMBER: Pyogenic abscess is always managed by aspiration/catheter drainage/liver resection as indicated with culture specific antibiotics. There is no role of only medical management in any case of pyogenic abscess)**

**Ans. Differential diagnoses of space occupying lesion in liver**

Non-neoplastic	Neoplastic			
	Benign		Malignant	
Hydatid cyst Amebic abscess Pyogenic abscess <i>Pneumocystis carinii</i> infection Hepatic TB Hepatic schistosomiasis	<b>Hepatocyte</b>	<b>Bile duct cell</b>	<b>Mesenchyme</b>	<b>Primary malignancy</b>
	Hepatocellular adenoma, Nodular regenerative hyperplasia, Focal nodular hyperplasia, Regenerative nodules	Hepatic cysts Cystadenoma Hamartoma Bile duct adenoma Polycystic liver disease	Hamartoma Lipoma Hemangioma Lymphangioma Hemangio-endothelioma Fibroma Leiomyoma	1. HCC Fibrolamellar HCC Clear cell carcinoma Carcinosarcoma Hepatoblastoma 2. Cholangiocarcinoma Cystadenocarcinoma 3. Angiosarcoma Leiomyosarcoma Lymphoma
				<b>Secondary Deposits</b>

**Management of a case of AMEBIC liver abscess**

Amebic liver abscess occurs when the organism reaches liver via the portal vein and causes liquefaction necrosis of liver parenchyma

[**Remember** : m.c. route of pyogenic abscess is through the biliary tree. Others include portal pyemia, direct contiguous spread, and hematogenous (hepatic artery)].

**Clinical presentation**

- The most common presenting symptoms are abdominal pain, dyspepsia and vomiting
- The most frequent sign is hepatomegaly. (**Right hypochondrium tenderness pyogenic**)
- Jaundice and fever can be present. Fever may suggest bacterial superinfection
- Incidence of amebic liver abscess is low in menstruating women
- Patients give a history of travel to endemic areas followed by diarrhea in recent past.

**Complications**

- Bacterial superinfection
- Free rupture into the peritoneal, pleural or pericardial cavities can occur.

**Diagnoses**

- TLC can be normal. Prolonged prothrombin time is usually seen.
- **Ultrasound** – Septae in the lesion are the biliary radicals which are intact while the rest of parenchyma is eaten up by liquefaction necrosis.
- CT scans.

**Management**

- In otherwise nonseptic patients, the treatment is purely medical
- Double dose **metronidazole** (750 mg tds/qid IV or 800 mg oral tds/qid) is started. Usually IV medication is given till patient is afebrile for 48 hours and then converted to oral to complete the 14 day course. Important side effects include metallic taste, peripheral neuropathy and disulfiram like reaction in alcoholics. It is contraindicated in pregnancy
- Monitoring of vitals – temperature, blood pressure, pulse and urine output is important

- Usually patient responds to the medical management and once 14 to 21 days antibiotic treatment is completed, it is followed by administration of luminal amebicide **Diloxanide fluroate** 500 mg tds for 10 days or **chloroquine** for 21 days can also be used. Paromomycin and Iodoquinol are other luminal amebicides.

**Indications of aspiration of amebic abscess are as follows:**

- Non resolution of symptoms after 4 to 5 days of antibiotic therapy
- Complications or contraindication of medical therapy
- Left lobe abscess or abscess in vicinity of vessels (hazardous location)
- Large abscess (> 5 to 6 cm) is also considered a relative indication for percutaneous aspiration
- Suspicion of pyogenic abscess
- Etiology not definitively diagnosed
- Impending rupture/ contained rupture of abscess.

These patients can be managed by percutaneous/open/laparoscopic aspiration/catheter drainage with antibiotics as mentioned above.

**Q48. Enumerate the important indications and contraindications of liver transplantation in present context.**

**Ans.**

**Indications of liver transplantation**

- **M.C. indication in children**—Biliary atresia
- **M.C. indication in adults**—Hepatitis C induced cirrhoses
- **M.C. indication in metabolic liver disease**—Alfa1 antitrypsin deficiency
- **M.C. indication in fulminant hepatic failure**—Acetaminophen toxicity
- **M.C. indication in auxilliary partial orthotopic liver transplantation**—Crigler Najjar syndrome

**List of indications of liver transplantation is as follows:**

**1. Cholestatic Liver Disease**

- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Biliary atresia
- Alagille syndrome

**2. Chronic Hepatitis**

- Hepatitis B
- Hepatitis C
- Autoimmune hepatitis

**3. Alcohol Liver Disease**

**4. Metabolic Diseases** such as Wilson disease, hemochromatosis, glycogen storage disease type 1 and 4, tyrosinemia, galactosemia, Niemann Pick disease, Gaucher disease, protein C/S deficiency, cystic fibrosis, protoporphyria, primary hyperoxaluria, Crigler Najjar disease, etc.

**5. Hepatocellular carcinoma**

**Milan criteria (Mazzaferro)**—Single tumor < 5 cm, 2 to 3 tumors none exceeding 3 cm, no vascular invasion or extrahepatic spread

**6. Neuroendocrine tumor metastases or colorectal tumor metastases in liver****7. Fulminant hepatic failure****8. Others**-polycystic liver disease, budd chiari syndrome, amyloidosis, cryptogenic cirrhosis.**Contraindications to liver transplantation****Absolute contraindications** to transplantation in recipient

- Active HIV with CD4 <200/cu mm
- Active uncontrolled systemic sepsis
- Severe comorbid cardiac/pulmonary disease/unreconstructable PVD
- Psychiatric/financial/compliance barriers
- Active drug abuse
- ABO incompatibility
- Extrahepatic malignancy

**Relative contraindications** to transplantation in recipient

- Controlled HIV infection
- Controlled but severe hypoxia (except due to hepatopulmonary syndrome)
- Hemodynamic instability
- Refractory psychiatric disorders
- Absence of adequate social support.

**Q49. Discuss grading and management of liver injury.**

**Ans.** Liver is the second most common organ injured in blunt trauma abdomen and penetrating trauma abdomen (after spleen in blunt trauma and after small bowel in penetrating trauma).

**Grading of liver injuries** is done in hemodynamically stable patients using CECT and is as follows:

Grade	Injury	Extent	Management Stable patient	Management Unstable patient
I	Hematoma Hematoma Laceration	Subcapsular < 10% surface area Parenchymal < 1 cm Capsular tear < 1 cm depth	Conservative	Surgery
II	Hematoma Hematoma Laceration	Subcapsular 10–50% Parenchymal 1–10 cm Capsular tear 1–3 cm depth	Conservative	Surgery
III	Hematoma Hematoma Laceration	Subcapsular > 50% / expanding/ ruptured Parenchymal >10 cm >3 cm depth	Conservative	Surgery
IV	Laceration	25–75% of hepatic lobe (1–3 segments in single lobe)	Angiography with/ out embolization	Surgery
V	Laceration	>75% lobe/>3 segments single lobe Juxtahepatic vena cava injury	Angiography with/ out embolization	Surgery
VI	Laceration	Hepatic arterial avulsion	Surgery	Surgery

- The trend has shifted towards conservative management of patients with liver trauma when they are hemodynamically stable especially in Grade 1, 2 and 3 trauma

- Hemodynamic stability is absence of tachycardia, hypotension, metabolic acidoses and no signs of shock at any time at presentation or after adequate resuscitation
- **In summary**, hemodynamic unstable patients go to the surgery directly. In the remaining patients, if the CT shows a grade IV or V injury, or a contrast extravasation, angiogram is recommended. If neither of these is noted, but the hemoglobin continues to decline “too quickly”, then also, an angiogram is warranted to rule out pseudoaneurysm.

**Complications of nonoperative management** include delayed rebleeding, pseudoaneurysm formation, hemobilia, and liver abscess.

**Options to manage liver injuries during surgery include**

- Perihepatic packing and manual compression
- Pringle maneuver (hepatoduodenal ligament control)
- Topical hemostatic agents or suture hepatorrhaphy
- Juxtahepatic vena cava injuries may require venovenous bypass and cardiovascular support for repair and is complex to manage.

## GALLBLADDER AND BILE DUCT

**Q50. Enumerate the risk factors for gallstone formation.**

**What are the types of gallstones? Discuss the causes of gallstone formation.**

**Write a note on pathophysiology of gallstone formation.**

**Ans.** The types of gallstones are as follows:

<b>Mixed stones</b>	
<b>Cholesterol stones</b>	
<b>Pigment stones</b> (< 30% cholesterol)	Black pigment stones (Calcium phosphate, bicarbonate and bilirubin pigment polymer) Brown pigment stones calcium palmitate, bilirubinate, stearate, cholesterol

**Causes and risk factors for gallstone formation are as follows:**

<b>Cholesterol stones</b>	<p><b>Cholesterol supersaturation</b> Obesity, increased dietary intake, clofibrate therapy, diabetes, rapid weight loss</p> <p><b>Decreased bile salts and lecithin</b> Ileal resection or bypass, cystic fibroses, pancreatic insufficiency,</p> <p><b>Nucleation</b> Bacterial infection of bile</p> <p><b>Stasis</b> High spinal injury, truncal vagotomy. Pregnancy, estrogen therapy, CBD stricture, prolonged TPN, prolonged fasting</p> <p><b>Genetic factors</b> Pima Indians (70%)</p>
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<b>Pigment stones</b>	Black	Old age Cirrhoses Hemolytic disorders Metabolic disorders
	Brown	Post cholecystectomy patients Biliary stents, parasites, sphincter of Oddi dysfunction

**Q51. Enumerate the complications associated with gallstones.****Discuss the spectrum of clinical presentations of cholelithiasis.**

**Ans.** Cholelithiasis can present as any of the following conditions and the clinical features can be written by memorizing the chart below:

<b>In gallbladder</b>	<ul style="list-style-type: none"> <li>• Asymptomatic stones</li> <li>• Biliary colic</li> <li>• Acute cholecystitis</li> <li>• Chronic cholecystitis</li> <li>• Xanthogranulomatous cholecystitis</li> <li>• Gallbladder cancer</li> <li>• Mirizzi's syndrome</li> </ul>
<b>Bile Duct</b>	<ul style="list-style-type: none"> <li>• Choledocholithiasis</li> <li>• Cholangitis</li> <li>• Pancreatitis</li> </ul>
<b>Intestine</b>	<ul style="list-style-type: none"> <li>• Gallstone ileus</li> </ul>

**Q52. Enumerate the types of cholecystitis. Discuss the management of a patient with acute calculous cholecystitis.****Ans.**

- Acute cholecystitis is an acute inflammation of the gallbladder
- Acute cholecystitis presents with clinical features like fever, pain and tenderness in right hypochondrium, Murphy's sign (where mid inspiration is inhibited by pain on palpation of the right upper abdomen quadrant), vomiting, or its complications including gallbladder perforation, gallbladder gangrene, emphysematous cholecystitis and empyema
- Although acute calculous cholecystitis is often considered an infection, bile cultures are positive only in 20 to 75% of patients. The organisms most commonly cultured are enteric bacteria including *Escherichia coli*, *Klebsiella*, and *Enterococcus*.

<b>Acute cholecystitis</b>	<b>Calculous Acalculous Xanthogranulomatous cholecystitis</b>
<b>Chronic cholecystitis</b>	Mucocele Uncomplicated
<b>Complicated cholecystitis</b> (Usually results from untreated acute cholecystitis)	Perforation (Acute, subacute, chronic) Empyema gallbladder Emphysematous Cholecystitis Cholecystoenteric fistula



**Diagnostic criteria of acute cholecystitis are given by the Tokyo guidelines** which are as follows:

**A. Local signs of inflammation**

(1) Murphy's sign, (2) RUQ mass/pain/tenderness

**B. Systemic signs of inflammation**

(1) Fever, (2) elevated CRP, (3) elevated WBC count

**C. Imaging findings: imaging findings characteristic of acute cholecystitis**

**Definite diagnosis**

- One item in A and one item in B are positive
- Confirms the diagnosis when acute cholecystitis is suspected clinically

**Note:** acute hepatitis, other acute abdominal diseases, and chronic cholecystitis should be excluded

**Ultrasonography findings of acute cholecystitis:**

- Sonographic Murphy sign
- Thickened gallbladder wall > 4 mm
- Enlarged gallbladder (long axis diameter > 8 cm, short axis diameter > 4 cm)
- Incarcerated gallstone, debris echo, pericholecystic fluid collection, sonolucent layer in the gallbladder wall, striated intramural lucencies, and Doppler signals.

**Tokyo guidelines classify severity of acute cholecystitis into three grades:**

*Mild (grade 1):*

Patients who do not meet the criteria of moderate or severe acute cholecystitis.

*Moderate (grade 2): Any one of the following conditions is present*

- WBC Counts > 18000/cu mm
- Palpable tender mass in right upper quadrant
- Duration of complaints > 72 hrs
- Marked local inflammation (biliary peritonitis, pericholecystic abscess, hepatic abscess, gangrenous cholecystitis, emphysematous cholecystitis).

*Severe (grade 3): Is cholecystitis with at least a single organ dysfunction.*

**Management guidelines according to Tokyo guidelines** have been formulated and are as follows:

*Medical treatment*

- NPO
- Intravenous fluids
- Antibiotics, and analgesia
- Close monitoring of blood pressure, pulse, and urinary output
- Simultaneously, the grade of severity needs to be established. Appropriate treatment should be performed in accordance with the severity grade.

Early cholecystectomy is recommended for most patients with laparoscopic cholecystectomy as the preferred method. Among high-risk patients, percutaneous gallbladder drainage is an alternative therapy for those patients who cannot safely undergo urgent/early cholecystectomy.

**Mild (grade I) acute cholecystitis**

Early laparoscopic cholecystectomy is the preferred treatment.

**Moderate (grade II) acute cholecystitis**

Early laparoscopic or open cholecystectomy is preferred. If a patient has serious local inflammation making early cholecystectomy difficult, then percutaneous or operative drainage of the gallbladder is recommended. Elective cholecystectomy can be performed after improvement of the acute inflammatory process.

**Severe (grade III) acute cholecystitis**

Management of severe local inflammation by percutaneous gallbladder drainage and/or cholecystectomy is needed. Biliary peritonitis due to perforation of the gallbladder is an indication for urgent cholecystectomy and drainage. Elective cholecystectomy may be performed after improvement of the acute illness by gallbladder drainage.

**Management of mucocele gallbladder**

Mucocele results from chronic accumulation of gallbladder secretions due to a stone at neck of gallbladder. It presents as a firm palpable gallbladder and features of cholecystitis. If not operated, and secondarily infected it can lead to empyema gallbladder. Late cases can also present as gallbladder perforation (Acute, Subacute or Chronic forms) and Emphysematous cholecystitis which are managed as discussed below.

Rupture of a true mucocele (malignant) can rarely result in pseudomyxoma peritonei.

**Surgery** – laparoscopic cholecystectomy is the treatment of choice.

Intraoperative decompression of gallbladder can be done if the distended gallbladder is difficult to grasp, but gross spillage should be avoided as in rare cases, it can be a true mucocele and spillage can lead to pseudomyxoma peritonei.

**Results and complications**

The reported mortality rate of acute calculous cholecystitis treated with laparoscopic cholecystectomy is less than 0.5% with reported morbidity rates of 5 to 20%.

A number of complications of acute calculous cholecystitis may occur including gallbladder perforation, gallbladder gangrene, emphysematous cholecystitis, and empyema.

The presence of any of these calls for urgent surgery.

- **Gangrenous cholecystitis** occurs in 2 to 30% of cases of acute calculous cholecystitis.
- **Perforation of the gallbladder** is present in 3 to 10% of patients with acute calculous cholecystitis, the most common form of perforation is subacute with walling off of the process as a pericholecystic abscess.

Spontaneous gallbladder perforation is of **three types** – acute presents with free biliary rupture and peritonitis. Subacute gets walled off as pericholecystic abscess and the chronic form is the biliary fistula.

- **Emphysematous cholecystitis** is an uncommon condition occurring when there is secondary infection of the gallbladder wall with gas-forming organisms. It is most common in elderly, diabetic men and it is treated with immediate administration of antibiotics (directed at anaerobes, coliforms, and clostridia species) and urgent surgery because of the high incidence of free perforation.

In all these complications, immediate management is tube cholecystostomy followed by stabilization of patients followed by definitive surgery in the form of cholecystectomy.

**Q53. Differentiate between medical and surgical jaundice.****Ans.**

Parameter	Medical Jaundice	Surgical/ Obstructive jaundice	Hepatocellular jaundice
Serum AST/ALT	N	Mild rise	High levels
Serum ALP/GGT	N	High levels	Mild rise/N
Total bilirubin	Mild rise	High rise	Moderate to high rise
Direct bilirubin	N	High rise	Mild rise/N
Urine bilirubin	Absent	High rise	Rise
Urine urobilinogen	High rise	Absent	Absent/N
Vander Waal test	1st stage reaction present	2nd stage reaction present	Can be both.

**Q. What are the causes of obstructive jaundice?****Classify the causes of surgical jaundice.****Ans. Causes of obstructive/surgical jaundice***Benjamin classification*

- Complete obstruction
  - Cholangiocarcinoma
  - Carcinoma head of pancreas
  - Carcinoma liver
  - Common bile duct ligation
- Intermittent obstruction
  - Duodenal diverticula (Lemmel syndrome)
  - Choledochal cyst
  - Polycystic liver disease
  - Choledocholithiasis
  - Biliary parasites
  - Hemobilia
  - Periampullary carcinoma
- Chronic incomplete obstruction
  - Stricture (congenital, iatrogenic, TB, post radiation, biliary enteric anastomotic stricture)
  - Primary sclerosing cholangitis
  - Chronic pancreatitis
  - Cystic fibrosis
  - Sphincter of oddi dysfunction
- Segmental obstruction
  - Traumatic
  - PSC
  - Cholangiocarcinoma
  - Intrahepatic stones
- Other causes – enlarged lymph nodes, hepatic artery aneurysm, Mirizzi's syndrome, postoperative (laparoscopy)

**Q. Discuss the pathophysiology of obstructive jaundice.**

**Explain the pathophysiological basis of the clinical features of surgical/ Obstructive jaundice.**

**Ans.**

**Pathophysiology**

- Normal CBD pressure – 7 to 14 cm water
- Biliary obstruction leads to increased CBD pressure
- At pressure >25 cm water, blood flow is decreased by 25% and diameter is decreased by 35%. This leads to cholangiolymphatic and cholangiovenous reflux
- At pressure 30 cm water, cholesterol and phospholipid secretion stops. Bile salt secretion also stops and bile becomes less lithogenic.

**Pathology**

- **Canaliculi** – dilatation and proliferation
- **Hepatocytes** – loss of microvilli, disrupted tight junctions between hepatocytes and biliary cells, bile thrombi in canaliculi, apoptosis of hepatocytes, decreased detoxification and synthetic function
- **Cholangiocytes** – atrophy, squamous metaplasia
- Cholangitis occurs only when obstruction is accompanied by increased intraductal pressure and infection.

**Other pathophysiological effects**

<b>Cardiovascular</b>	<ul style="list-style-type: none"> <li>• Decreased left ventricular contractility</li> <li>• Decreased extracellular volume</li> <li>• Decreased peripheral resistance</li> <li>• Hypotension is the final result</li> </ul>
<b>Renal</b>	<ul style="list-style-type: none"> <li>• Decreased tubular function due to peritubular deposition</li> <li>• Direct natriuretic, kaliuretic, diuretic effect</li> <li>• Direct parenchymal damage</li> <li>• Decreased cardiac function also affects</li> </ul>
<b>Coagulopathy</b>	<ul style="list-style-type: none"> <li>• Increased prothrombin time (m.c.)</li> <li>• Decreased vit. K, calcium</li> <li>• Endothelial damage</li> </ul>
<b>Immunity and wound healing</b>	<ul style="list-style-type: none"> <li>• Decreased cell mediated immunity</li> <li>• Decreased kupffer cell function</li> <li>• Bacterial translocation</li> <li>• Decreased antiendotoxic effect of bile salts in small intestine</li> <li>• Decreased propylhydroxylase activity in skin</li> </ul>
<b>Anorexia</b>	<ul style="list-style-type: none"> <li>• Due to lack of bile acids in intestine</li> </ul>

**Q. Discuss the diagnostic approach in a case of obstructive jaundice and explain the basis of preoperation preparation of jaundiced patient.**

**Write a note on management of a patient with obstructive jaundice.**

**Ans. Diagnoses**

*History*

- Age—older age more towards malignancy

- Sex—primary biliary cirrheses, choledochal cyst is more common in females
- Symptoms—painless/painful jaundice, pruritus, melena, anorexia, weight loss, vomiting, hematemesis, episodes of dyspepsia, abdominal mass
- Past history—diabetes (in 25% of patients with carcinoma pancreas)  
Past biliary surgery, blood transfusion
- Family history of jaundice, hereditary spherocytosis, carcinoma personal history of smoking, alcohol, drugs/injections history.

#### *Examination*

- General examination – encephalopathy, hypotension, pallor, jaundice, clubbing
- Left supraclavicular lymphadenopathy, blummershelf
- Scratch marks due to itching
- Migratory thrombophlebitis
- Signs of liver cell failure
- Ascites, right hypochondrium mass, palpable gallbladder, liver, spleen
- Rectal primary/hemorrhoids on per rectal examination might be present.

#### **Investigations**

Biochemical tests to establish the presence of obstructive/surgical jaundice

- Liver function test—serum bilirubin with direct and indirect components, ALT, AST, ALP, GGT, total protein and albumin, coagulation parameters (PT, aPTT, INR)
- Total and differential leucocyte counts, hemoglobin, platelet count, electrolytes and sugar
- Urine urobilinogen and bilirubin
- Stool for occult blood/ steatorrhea/absence of bile pigments.

#### **Imaging work up**

- **Trans-abdominal ultrasound** to get some clue as to the cause of obstructive jaundice as given by intrahepatic or extrahepatic biliary dilatation.

*To know the site and level of obstruction, we then move towards specific tests*

- **MRCP** is the non-invasive investigation of choice for this purpose.
- **EUS** through side viewing endoscope or ERCP with EUS are invasive modalities of choice for this purpose.
- **CT cholangiogram** can also be useful in this regard.

**Tumor marker levels**—CEA, transthyretin, Ca 19 to 9 levels can be measured to support imaging findings and establishing a diagnoses

Other rare tests useful in the work up of a patient with obstructive jaundice include:

- **Technetium-99m labeled RBC scan or angiography** for hemobilia
- **Upper GI series** for duodenal webs or diverticula
- **PTC** for iatrogenic biliary strictures or leaks.

A variety of combination of these tests help in identifying most cases of obstructive jaundice.

#### **Preoperative optimization of jaundiced patient**

- Nutrition and hypoproteinemia need to be taken care of. However, care should also be taken to avoid protein overload too as this can precipitate hepatic encephalopathy
- Fluid and electrolyte management

- Carbohydrate replacement
- Anemia correction
- Correction of coagulopathy—vitamin K daily for 5 days, FFP and cryoprecipitates if no response to vitamin K
- Chest physiotherapy as patient is going to undergo upper abdominal surgery
- Infection control (antibiotics)
- DVT prophylaxis as indicated for the patient
- **Preoperative biliary stenting** only in cases with
  - Nonresolving cholangitis over 24 hours
  - Comorbidities delaying operation
  - Severe coagulopathy in the patient.

**Q54. Enumerate the complications after laparoscopic cholecystectomy.**

**Write a note on postcholecystectomy problems.**

**Ans.**

**Complications of laparoscopic cholecystectomy are as follows:**

Due to general anesthesia	Due to laparoscopy	Due to cholecystectomy
<ul style="list-style-type: none"> <li>• Oral/teeth trauma</li> <li>• Sore throat</li> <li>• Nausea/Vomiting/Headaches</li> <li>• Myalgia/paresthesia</li> <li>• Psychosis/transient amnesia</li> <li>• Anesthesia awareness</li> </ul>	<ul style="list-style-type: none"> <li>• Intraoperative or post-operative bleeding</li> <li>• Trocar injury</li> <li>• Port site complications – Bleed, Infection, Hernia</li> <li>• Atelectasis, pneumonia, pneumothorax,</li> <li>• Bradycardia</li> </ul>	<ul style="list-style-type: none"> <li>• Bowel injury (Hepatic flexure)</li> <li>• Liver injury</li> <li>• Post cholecystectomy problems (Discussed below)</li> <li>• Port site metastasis</li> </ul>

**Post cholecystectomy syndrome**

Persistence of pain and other pre-operative symptoms after laparoscopic cholecystectomy regardless of etiology is called post-cholecystectomy syndrome.

*Causes*

Biliary	Nonbiliary
Biliary leak [minor or major (>500 ml/24hrs)]	Pancreatic cause
Hematoma/abscess/dropped stones	Peptic ulcer/GERD
Mechanical causes – stricture/fistula/malignancy/retained stone (< 2 years of surgery)	Irritable bowel syndrome
Port site hernia/neuroma	Diverticular disease
Sphincter of Oddi disorders	Psychosomatic illness

**Bile Duct Injury**

Most common cause – iatrogenic – Laparoscopy > open cholecystectomy

*Risk factors*

- Inflammation in the porta hepatis
- Variable biliary anatomy

- Inappropriate exposure
- Aggressive attempts at hemostasis
- Surgeon factors (visual misperception > technical skill or knowledge)

Cholangiography does not completely avoid bile duct injury, but may reduce the incidence and extent of injury.

#### *Classification*

- Strasberg – Bismuth classification of bile duct injuries

Type A	Cystic duct stump leak or leak from small injuries near liver bed	
Type B	Transection and occlusion of a part of biliary tree especially aberrant right hepatic duct	
Type C	Transection without occlusion of aberrant right hepatic ducts	
Type D	Lateral injuries to major bile duct	
Type E	E1	Common hepatic duct division > 2 cm from bifurcation
	E2	Common hepatic duct division < 2 cm from bifurcation
	E3	Division at bifurcation but intact confluence
	E4	Disruption of bifurcation and separation of right and left system
	E5	Involvement of aberrant right hepatic duct with/out CHD stricture.

#### **Stewart way classification of laparoscopic bile duct injuries**

- **Class I:** Common bile duct (CBD) is mistaken for the cystic duct, but the error is recognized before the CBD is divided.
- **Class II:** Damage to the common hepatic duct from clips or cautery used too close to the duct especially in cases where visibility is limited due to inflammation or bleeding.
- **Class III (most common type):** CBD is mistaken for the cystic duct. The common duct is transected and a variable portion that includes the junction of the cystic and common duct is excised (removed).
- **Class IV:** Damage to the right hepatic duct (RHD), either because this structure is mistaken for the cystic duct or is injured during dissection.

#### *Clinical Presentation*

- Identified intraoperatively or in the postoperative period
- Bile peritonitis
- Bile duct stricture and jaundice – upto 70% of strictures present within 6 months of the surgery.

#### *Treatment*

Recognized at the time of cholecystectomy

- Conversion to an open operation
- Use of cholangiography
- Injury of duct < 3 mm—simple ligation
- Injury of duct > 3 mm—re-implantation into the biliary tree
- Large duct injury except cautery injuries and less than 50% circumference – T-tube and primary repair

- Cautery injury of large duct or > 50% circumference involved – Biliary enteric reconstruction or biliary to biliary reconstruction (if injury is near hepatic duct.)– choledochoduodenostomy or Roux-en-Y chledochojejunostomy reconstruction with T-tube stenting is the preferred procedure.

Identified after cholecystectomy

- Symptoms of biloma/biliary fistula/ biliary ascites/ biliary stricture prompt towards this diagnoses in the postoperative period after laparoscopic or open cholecystectomy
- Imaging techniques
- First test to be performed after USG—CECT abdomen
- Best test—HIDA scan > PTC
- Goals of therapy in Iatrogenic bile duct injury
  - **Control of infection and limiting inflammation with help of** parenteral antibiotics and percutaneous drainage of periportal fluid collections
  - **Complete delineation of entire biliary anatomy with help of** MRCP/PTC or ERCP (especially if cystic duct stump leak suspected)
  - **Re-establishment of biliary enteric continuity with** tension-free, mucosa-to-mucosa/Roux-en-Y hepaticojejunostomy/long-term transanastomotic stents if involving bifurcation or higher.

*Independent factors associated with stricture recurrence include:*

- Primary repair within 3 weeks of the initial injury
- The number of attempts at repair is inversely correlated
- Cholangitis prior to repair
- Incomplete cholangiography
- The use of transanastomotic stents is associated with a favorable outcome
- Chronic liver disease and hepatic fibrosis are associated with higher operative mortality and lower success rates.

### **Lost Stones**

- Can lead to chronic intraabdominal abscess, fistula, wound infection and bowel obstruction
- Treatment - Extensive irrigation, significant attempt to retrieve lost stones and antibiotics.

### **Retained Biliary Stones**

Endoscopic removal of these stones via a sphincterotomy.

### **Biliary Leak**

- Patients will generally present within 1 week of cholecystectomy
- CECT will show ascites or a right upper quadrant fluid collection consistent with a biloma
- Endoscopic cholangiography should be performed with percutaneous drainage of any fluid collections
- If the leak is from a cystic duct stump, sphincterotomy with stenting of the common duct
- Re-exploration for the patients with septic shock or failure of percutaneous procedure or inaccessible location for percutaneous procedure. Definitive surgery should be withheld till the inflammation subsides like in bile injury settings and preferably done after 4 to 6 weeks.



**Q55. Write a note on choledochal cyst.**

**Mention the clinical features and discuss the management of a patient with choledochal cyst.**

**Ans. Pathogenesis**

Presence of an anomalous pancreaticobiliary junction (APBDJ) leads to reflux of pancreatic secretions into biliary duct and causes cystic degeneration.

**Todani modification of Alonso-Lej classification**

Type	Description	
I	A	Cystic dilatation of extrahepatic bile duct
	B	Fusiform dilatation of extrahepatic bile duct
	C	Saccular dilatation of extrahepatic bile duct
II	Choledochal diverticulum	
III	Choledochoce (Intraduodenal choledochal diverticulum)	
IV	A	Intrahepatic and extrahepatic duct cysts
	B	Multiple cysts in extrahepatic ducts only
V	Multiple cysts in intrahepatic ducts only (caroli disease)	
VI	Cystic dilatation of the cystic duct alone	

**Presentation**

- More common in women
- More common in patients of asian descent
- More commonly present in infancy
- Jaundice (most common symptom in adults), right upper quadrant pain and a palpable mass
- Nausea, pruritus, weight loss
- Cirrhosis, cholangitis, pancreatitis, hepatic fibrosis and malignancy
- Acute rupture of the cyst and bile peritonitis
- As cholangiocarcinoma (10 to 30% incidence)

**Diagnoses**

- CECT for diagnoses, MRCP for characterization and ERCP for identification of APBDJ.

**Surgical management**

- Resection of the entire cyst and appropriate surgical reconstruction
- Type I cysts complete surgical excision, cholecystectomy and Roux-en-Y hepaticojejunostomy
- Type II cysts excision and biliary enteric diversion by Roux-en-Y hepaticojejunostomy
- Type III cysts—endoscopic drainage, transduodenal excision or sphincteroplasty
- Type IV B cysts—Similarly to type I cysts with excision and hepaticojejunostomy
- Type IV A with only one lobe involved can be treated with partial hepatectomy and reconstruction
- Caroli's disease—Resection if the disease is unilobar to liver transplantation when diffuse disease is detected.

It should however be remembered that resection does not eliminate the risk of malignancy.

**Q56. Write a note on hemobilia and bilhemia.**

**Ans.** Defined as bleeding into the biliary tree from an abnormal communication between a blood vessel (portal vein or hepatic artery) and bile duct.

**Common causes**

- Iatrogenic trauma (m.c.) (PTBD>PTC)
- Accidental trauma (Blunt > Penetrating)
- Gallstones (cholecystitis/mechanical trauma by stones)
- Tumors (Liver, gallbladder, biliary tree and pancreas)
- Inflammatory disorders (parasitic infections, hepatic abscesses, cholangitis)
- Vascular disorders (aneurysms, angiodyplasia, hemangioma).

**Clinical Presentation**

- **Sandblom triad**
  - Upper abdominal pain
  - Upper gastrointestinal hemorrhage (melena > hematemesis)
  - Jaundice
- Clots can cause cholangitis, pancreatitis and cholecystitis.

**Diagnostic workup**

- Upper gastrointestinal endoscopy
- If upper endoscopy is diagnostic and conservative management is planned, no further studies are necessary
- Contrast-enhanced CT—pooling contrast, intraluminal clots or biliary dilation, cavitating central lesions and aneurysms
- Arterial angiography—Test of choice.

**Treatment and outcomes**

- Stopping the bleeding and relieving biliary obstruction
- Correction of coagulopathy
- The first line of therapy for major hemobilia is transarterial embolization (TAE)
- Surgery is indicated when conservative therapy and TAE have failed
  - Procedures involve ligation of bleeding vessels, excision of aneurysms, or nonselective ligation of a main hepatic artery
- Hepatic resection may be necessary for failed arterial ligation or for cases of severe trauma or tumor
- Hemorrhage from the gallbladder or hemorrhagic cholecystitis mandates cholecystectomy. Endoscopic coagulation, somatostatin and vasopressin.

**Bilhemia**

- Bilhemia is a condition in which bile flows into the bloodstream through the hepatic veins or portal vein branches
- Occurs in presence of a high intrabiliary pressure exceeding that of the venous system
- Causes—Gallstones eroding into the portal vein or accidental or iatrogenic trauma

- The clinical presentation is that of rapidly increasing obstructive jaundice without elevation of hepatocellular enzyme levels (e.g. AST, ALT) and septicemia
- This diagnosis is best determined by ERCP
- Treatment—Lowering intrabiliary pressures through stents or sphincterotomy.

**Q57. Write a note on management of choledocholithiasis.**

**What are the types of CBD stones? Discuss its clinical features and management.**

**Ans.**

<b>Primary CBD stones</b>	<ul style="list-style-type: none"> <li>• Arise denovo in CBD</li> <li>• Asian origin people are predisposed</li> <li>• Are brown stones</li> <li>• Bacterial infection predispose. Therefore also called infection stones</li> </ul>
<b>Secondary CBD stones</b>	<ul style="list-style-type: none"> <li>• Come from gallbladder into CBD</li> <li>• US origin are predisposed</li> </ul>
<b>Retained CBD stones</b>	Found within 2 years of cholecystectomy
<b>Recurrent CBD stones</b>	After 2 years of cholecystectomy
<b>Silent CBD stones (10% incidence in intraoperation cholangiography and 0.2-5% in postoperative period)</b>	Identified during cholangiography performed for other reason

**Clinical manifestation**

- Charcot's triad
  - Biliary colic
  - Obstructive jaundice
  - Fever
- Hypotension and mental status changes with Charcot's triad is known as **Reynolds pentad**.

**Diagnosis**

- **Ultrasound**
- **ERCP** is highly sensitive and specific and can usually be **therapeutic** by clearing the duct of all stones in approximately 75% of patients during the first procedure and approximately 90% with repeat ERCP
- **Indications for preoperative ERCP** prior to cholecystectomy include cholangitis, biliary pancreatitis, limited surgeon experience with common duct exploration and patients with multiple comorbidities
- **MRCP** is highly sensitive (>90%) with an almost 100% specificity for the diagnosis of common duct stones and is noninvasive but not therapeutic
- **PTC** can also be used to diagnose and treat choledocholithiasis
- **Dilated CBD is when > 6 mm (USG), > 8 mm (MRCP), >10 mm (ERCP), > 12 mm (external diameter in open surgery).**

**Treatment**

- Preoperative diagnoses—ERCP and stone extraction with/out endoscopic sphincterotomy

- Intraoperative suspicion—Do intraoperative cholangiography
- Intraoperative confirmation of small stone (<4–6 mm)—Glucagon instillation can aid in removal
- Intraoperative confirmation during laparoscopic cholecystectomy—Laparoscopic transcystic or choledochotomy and stone removal followed by check cholangiogram OR conversion to open procedure and open CBD exploration with primary closure over T-tube or biliary enteric anastomosis (choledochoduodenostomy or Roux-en-Y choledochojejunostomy)
- Postoperative determination of CBD stones—ERCP and stone extraction and endoscopic sphincterotomy.

**Contraindications for the transcystic approach**

- Small, friable cystic duct
- Numerous (more than eight) stones in the common bile duct
- Large stones (>1 cm)
- Stones in CHD above the cystic duct insertion

**Indications of choledochotomy**

- Palpable CBD stones
- Cholangitis with obstructive jaundice
- CBD > 12mm
- Demonstration of stone on intraoperative cholangiogram

Most common complication of laparoscopic CBD exploration—Retained Stone (5%)

**Contraindications of ERCP**

- Low lying biliary stricture
- Impacted stones/multiple stones
- Stone > 1.5 cm
- Intrahepatic stones.

**Contraindications of endoscopic sphincterotomy**

- Severely inflamed duodenal wall and head of pancreas
- Long suprasphincteric stricture > 15 mm
- CBD diameter > 2 cm
- Perivaterian diverticulum

**Ideal for open CBD exploration and reconstruction**

- CBD > 1.2 cm (preferably >2cm)
- No duodenal ulcer
- No pancreatitis

**Plan of action when ERCP is contraindicated or fails**

- Supraduodenal choledochotomy/transduodenal sphincteroplasty is the next best option
- Choledochoduodenostomy/Roux-en-Y reconstruction hepaticojejunostomy is the last best option.

**Q58. Enumerate the causes of Biliary Strictures.****Ans.**

<b>Postoperative</b>	<ul style="list-style-type: none"> <li>• Laparoscopic or open cholecystectomy</li> <li>• CBD exploration</li> <li>• Recurrence after previous stricture repair</li> <li>• Gastrectomy, hepatic resection/transplantation</li> <li>• Pancreatic surgery</li> <li>• Biliary enteric anastomotic stricture</li> </ul>
<b>Biliary tree intervention</b>	<ul style="list-style-type: none"> <li>• Percutaneous or endoscopic</li> </ul>
<b>Benign causes</b>	<ul style="list-style-type: none"> <li>• Primary sclerosing cholangitis</li> <li>• Chronic pancreatitis, choledocholithiasis</li> <li>• Mirizzi's syndrome</li> <li>• Duodenal diverticulum</li> <li>• Radiation fibrosis</li> <li>• Parasitic biliary infections</li> <li>• Subhepatic abscess</li> <li>• Crohn's disease</li> </ul>
<b>Congenital causes</b>	<ul style="list-style-type: none"> <li>• Web, stricture</li> <li>• Choledochal cyst</li> <li>• Caroli disease</li> <li>• Biliary atresia</li> </ul>
<b>Malignant causes</b>	<ul style="list-style-type: none"> <li>• Cholangiocarcinoma</li> <li>• Pancreatic carcinoma</li> <li>• Periampullary carcinoma</li> </ul>

**Q59. What is Courvoisier's law? Write its fallacies.**

**Ans.** Courvoisier law states that in the presence of jaundice, an enlarged gallbladder is unlikely to be due to gallstones, carcinoma of the pancreas, duodenum, gallbladder or common bile duct and lymph node metastasis in porta hepatis is more likely.

The reason is that the gallbladder gets shrivelled and chronically fibrosed in patients with longstanding gallstones and therefore cannot be enlarged.

**Exceptions to this rule:** That is, enlarged gallbladder in presence of gallstones can be in one of the following circumstances:

- Double impaction—one in cystic duct and one in CBD
- Oriental cholangiohepatitis
- Pancreatic calculus obstructing the ampulla of Vater
- Mucocele due to stone impacted in the cystic duct.

## PANCREAS

**Q60. Write a note on annular pancreas.****Ans. Embryology**

The pancreas arise from two anlage. Ventral anlage from the hepatic diverticulum and the dorsal anlage from the dorsal mesogastrium. The ventral anlage rotates around the duodenum to fuse with the dorsal anlage. The duct of the ventral anlage (posterior aspect

of head and uncinate process) is the duct of Wirsung and the duct of dorsal anlage (anterior aspect of head, neck, body and tail of the pancreas) is the duct of Santorini. Normal pancreas drain through the duct of Wirsung which opens into the major papilla after fusion with main part of duct of Wirsung and the remaining part of duct of Santorini opens into the minor papilla.

**(ALWAYS REMEMBER:** The most common congenital anomaly of the ductal system is the pancreas divisum, i.e. nonfusion of the duct of Wirsung and Santorini. The result is most of the pancreas drain through the duct of Santorini and the minor papilla. This predispose to attacks of acute pancreatitis and the treatment for this acute pancreatitis is minor papillary sphincterotomy.)

Annular pancreas result when the ventral pancreatic anlage fail to rotate around the duodenum completely. This can result in two types of annular pancreas

- **Extramural type**—Ventral pancreatic duct encircles to join the duodenum
- **Intramural type**—Pancreatic tissue is intermingled with the muscle fibers of the duodenum and all open directly into the duodenum.

Theories of occurrence of annular pancreas

- **Lecco's theory**—Adhesion of the ventral anlage to the duodenal wall before rotation
- **Baldwin theory**—Persistence of the left ventral anlage
- **Third theory**—Tip of the left ventral anlage adheres to duodenum and persists when the duodenum rotates and results in a ring of pancreatic tissue.

**“YOGI”** Classification system of annular pancreas has 6 types with type 1 being the most common type in which the annular duct joins the main pancreatic duct drains into the major papilla.

### Clinical features

- Incidence – 1 in 20,000 births
- Isolated finding or associated with duodenal atresia, stenosis, web
- In children manifest as duodenal or bile duct obstruction or recurrent pancreatitis
- In adults, manifests as duodenal obstruction, chronic pancreatitis or peptic ulcer disease
- Most common manifestation in adults is epigastric pain.

### Investigations

- **MRCP is the investigation of choice**
- **Crocodile jaw sign** suggests annular pancreas

### Treatment

- **Duodenoduodenostomy is the treatment of choice**
- No attempt should be made to cut the ring of annular pancreas
- Rest management is same as that for a patient with acute pancreatitis.

## Q61. Define acute pancreatitis and enumerate its causes.

**What is acute pancreatitis. What are the causes of acute pancreatitis.**

**Ans.** According to the **revised Atlanta classification** (2012), the acute pancreatitis is defined as presence of two of the following three features:

- Abdominal pain characteristic of pancreatitis (Epigastric pain radiating to the back)

- Serum amylase and lipase levels greater than 3 times the normal
- Characteristic findings on CECT, MRI or USG studies.

Pancreatitis is divided into 2 phases – **Early** (1st week) and **Late** (2nd week onwards)

**Severe acute pancreatitis** is defined in the early phase as any organ failure lasting for greater than 48 hours or death and during the second phase as persistent organ failure or complications resulting from acute pancreatitis or death. All other cases are called **mild acute pancreatitis**.

**Causes of acute pancreatitis** are as follows (MN – Gate has mets in cap)

- Gallstones, alcohol, trauma, ERCP
- Hereditary pancreatitis, hypertriglyceridemia, hypercalcemia
- Sulfonamides, azathioprine, mercaptopurine, estrogens, tetracycline
- Sphincter of Oddi dysfunction
- Infections (Mumps, cytomegalovirus, coxsackie virus, echovirus, parasites)
- Neoplasm of pancreas
- Autoimmune (Sjogren's syndrome)
- Cystic fibrosis, pancreas divisum, peripapillary diverticulum.

**Q. Write a note on clinical features and management of acute pancreatitis.**

**Discuss the management outline of a patient of acute pancreatitis.**

**Ans. Clinical features**

*History*

- Epigastric pain radiating to back, increases with supine posture or food intake and relieved on analgesic or on bending forward
- Nausea, anorexia, vomiting
- Fever might be present due to systemic response or infected necrosis
- Muscle spasms or other features of hypocalcemia
- Dyspnea, cough, chest pain due to pleural effusion
- Abdominal distension, obstipation due to ileus or ascites

*Examination*

- General examination may reveal altered consciousness, dehydration, tachycardia, tachypnea, hypotension, elevated temperature and decreased urine output.
- Abdominal examination may reveal abdominal distension on inspection and epigastric tenderness and other features of acute abdomen on palpation. Bowel sounds can be absent due to associated ileus.

Prognostic scoring of the patient should be started as soon as the patient is suspected to have acute pancreatitis and patient investigated accordingly.

**Complications of acute pancreatitis/phases of acute pancreatitis are as follows:**

- Local

Duration	Type of pancreatitis	Type of collection
< 4 weeks duration	Interstitial edematous pancreatitis (IEP)	Sterile or infected acute peripancreatic fluid collection

*Contd...*

Contd...

Duration	Type of pancreatitis	Type of collection
	Necrotizing pancreatitis	Sterile or infected pancreatic and/or peripancreatic acute necrotic collection
>OR = 4 weeks duration	Interstitial edematous pancreatitis (IEP)	Sterile or infected pancreatic pseudocyst
	Necrotizing pancreatitis	Sterile or infected walled off necrosis
Other local complication		Splenic vein thrombosis or splenic artery pseudoaneurysm

**Always remember :** There is no term as 'pancreatic abscess' now.

*Systemic complications [More common in the first week]*

- Hyperglycemia, hypocalcemia, hyperlipidemia
- DIC, Renal failure
- **CNS** -Confusion, encephalopathy, transient visual disturbances (**Purtscher's Retinopathy**)
- **Pulmonary** - ARDS, atelectasis, pleural effusion, pneumonia
- **Cardiac** – Pericardial effusion, arrhythmias, shock
- Intra-abdominal saponification, subcutaneous fat necrosis

### Investigations

**Blood** - TLC, Hb, Platelet count and PT/ PTTK/ INR, serum amylase, lipase, calcium – both ionized and free, serum electrolytes (Na, K, Mg), liver and kidney function test, albumin levels, glucose levels.

### *Radiological investigations*

Ultrasound abdomen is the first investigation usually done. If the patient is diagnosed based on the first two points of the definition and he has a mild attack, no radiological investigation is absolutely necessary.

CECT abdomen is the investigation of choice as it helps in diagnoses, identification and staging of the local complications and prognostic scoring of the acute pancreatitis. MRCP, ERCP, EUS can also be used as per the stage and complication of the pancreatitis.

### Treatment

#### *Mild acute pancreatitis*

Conservative management as follows:

- Bowel rest – Nil per oral, nasogastric tube placement, feeding by postpancreatic means such as feeding jejunostomy or nasojejunal tube
- Temperature, Intake – output, pulse, blood pressure, respiratory rate and SpO<sub>2</sub> monitoring
- IV fluid supplementation as required and electrolyte and acid base balance to be maintained
- If gallstones are the cause then early cholecystectomy within 24 to 72 hours is the treatment of choice and can be performed laparoscopically.

#### *Severe pancreatitis*

- To be managed in ICU setting with above measures as well as the following additional measures



- CECT to be done after 48 to 72 hours of admission if the suspicion is of severe pancreatitis. NCCT is done in emergency for confirmation of diagnoses
- If patient has high grade fever, tachycardia, and other features of sepsis, than CECT for identifying infected necrosis (presence of gas in pancreatic or peripancreatic tissue) is seen and if in doubt, FNAC should be done to confirm the presence of infected necrosis
- **Sterile necrosis** is managed with conservative measures as mentioned above
- **Infected necrosis** is managed with starting antibiotics (Imipenem, Meropenem, Ciprofloxacin) alongwith percutaneous aspiration or catheter drainage. Surgery has a prohibitive mortality rate in first 4 weeks and should be withheld as long as possible (**Step Up approach of PANTER trial** which means least invasive to most invasive measures used in sequence). The cholecystectomy here is done after minimum 6 weeks with preoperative ERCP in most cases to rule out CBD stones if it is a case of gallstones.

#### Q62. What is pseudocyst pancreas? What are its causes?

**Discuss the clinical features, complications and management of pseudocyst pancreas.**

**Ans.** Acute fluid collection in a patient with acute interstitial edematous pancreatitis with duration more than 4 weeks or in the patients with chronic pancreatitis and lined by fibrogranulation tissue is called pseudocyst pancreas.

(**ALWAYS REMEMBER:** Duration should be more than 4 weeks. Pancreas or peripancreatic tissue should not have necrosis.)

#### Causes

- Acute interstitial edematous pancreatitis
- Chronic pancreatitis (Alcohol > Gallstones) – **m.c. cause in adults**
- Trauma abdomen (Blunt > penetrating trauma)- **m.c. cause in children.**

#### Site

- m.c. site – lesser sac
- can occur anywhere from mediastinum to scrotum

#### Clinical features

- Present as acute pancreatitis or chronic pancreatitis (Exocrine or endocrine ion sufficiency)
- Anorexia, nausea, vomiting, and features of **gastric outlet obstruction**
- **With complications of pseudocyst** – Infected pseudocyst, rupture, hemorrhage into pseudocyst, malignancy in the pseudocyst enteric fistula, vascular complications such as splenic artery pseudoaneurysm and splenic vein thrombosis.

#### Investigations

- CECT pancreas protocol is the investigation of choice. ERCP > MRCP is done for looking for the presence of ductal communication (which is present in only 10% of patients)
- Follow up of the patients planned for conservative management is done using ultrasound.

#### Treatment

- Asymptomatic uncomplicated—conservative management for up to 6 to 8 weeks
- Alcohol abstinence

- Symptomatic/complicated/size > 8 to 10 cm—Endoscopic cystogastrostomy or surgical open/laparoscopic cystojejunostomy or cystogastrostomy or cystoduodenostomy with mandatory biopsy of the cyst wall to rule out malignancy and management of underlying acute or chronic pancreatitis and its cause.

**Q63. Write a note on Insulinoma.**

**Ans.**

- Most common functioning tumor of the endocrine pancreas
- Equal distribution in the head, body and tail.

**Clinical features**

- Small tumors with an average size of 1.0 to 1.5 cm.

**Whipple's triad**

- Fasting-induced neuroglycopenic symptoms of hypoglycemia (diaphoresis, shaking, mental confusion, obtundation and seizures)
- Low blood glucose levels (40 to 50 mg/dL)
- Relief of symptoms after the administration of glucose.

Patients with insulinomas often report a significant weight gain associated with the onset of symptoms as they compensate by eating frequently to prevent hypoglycemia.

**Diagnoses**

- **72-hour fast** - Insulin, glucose, proinsulin, and C peptide levels are measured every 6 hours until the glucose level is lower than 60 mg/dL and then every 1 to 2 hours or until the patient becomes symptomatic
  - 75% of patients have test positive within 24 hours and 95% by 72 hours
  - Serum insulin (>5  $\mu$ U/mL) in the setting of hypoglycemia
  - Insulin-to-glucose ratio is higher than 0.3 occurs with insulinoma [ $\mu$ U/mL of insulin/ (mg/dL of glucose)]
  - C peptide levels higher than 1.2  $\mu$ g/mL with a glucose level lower than 40 mg/dL.
- **Localization**
  - CT or MRI should be performed
  - Hyperattenuating on contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI)
  - If not, EUS should be performed
  - If the tumor remains unlocalized, angiography with or without stimulation should be performed. If all of the above being negative blind exploration and intraoperative ultrasound be performed.
  - **Show a vascular blush in angiography**
  - **SRS is not useful for insulinomas.**

**Treatment**

- Surgical resection
- Preoperatively glucose infusions must be used in the perioperative period, especially when patients are taking nothing by mouth. Administration of diazoxide decreases

beta cell release of insulin (usually 3 mg/kg/day, divided into two or three daily doses)

- Enucleation is the preferred treatment.

### Contraindication

- If the tumor is within 2 mm of the main pancreatic duct.

In these cases,

- Resection via distal pancreatectomy, central pancreatectomy, or pancreaticoduodenectomy may be necessary for tumors abutting the main pancreatic duct or for large tumors
- MEN1 patients may require a combination of partial pancreatic resection—distal pancreatectomy or pancreaticoduodenectomy—and enucleation for multiple lesions in the pancreas.

### Q64. Write a note on prognostic scoring in pancreatitis patients (severity scoring).

Ans. **A. Ranson score (For non gallstone induced pancreatitis) (TOTAL - 11)**

<b>On Admission</b>	<ul style="list-style-type: none"> <li>• Age &gt; 55 years</li> <li>• TLC &gt; 16,000</li> <li>• Blood glucose &gt; 200 mg/dl</li> <li>• LDH &gt; 350 IU/L</li> <li>• AST &gt; 250 IU/L</li> </ul>
<b>After 48 hours of admission</b>	<ul style="list-style-type: none"> <li>• Base deficit &gt; 4</li> <li>• BUN elevation &gt; 5</li> <li>• Fluid sequestration &gt; 6 litre</li> <li>• Calcium fall &gt; 8</li> <li>• Hematocrit fall &gt; 10</li> <li>• Arterial PO<sub>2</sub> &lt; 60 mm Hg</li> </ul>
<b>Prognostication</b>	<ul style="list-style-type: none"> <li>• &lt;3—1-2% mortality</li> <li>• 3-4—15% mortality</li> <li>• 5-6—30% mortality</li> <li>• 7 or more—50-90% mortality</li> </ul>

In case of gallstone induced pancreatitis, changes in Ranson score include

Age > 70 years, TLC > 18,000, sugar > 220 mg/dl, LDH > 400 IU/L, sequestration > 4 lit. BUN elevation > 2. Base deficit > 5. Rest are same.

### B. Modified Glasgow criteria

*Evaluated during initial 48 hours*

<b>Age</b>	>55 years
<b>TLC</b>	>15,000/cu.mm
<b>Glucose</b>	> 180 mg/dl
<b>LDH</b>	>600 IU/L
<b>PO<sub>2</sub></b>	<60 mm Hg
<b>Calcium</b>	<8 mg/dl
<b>Albumin</b>	<3.3 g/dl
<b>BUN (blood urea nitrogen)</b>	>45

Value > 2 suggest severe pancreatitis and high mortality rate.

**C. Apache 2 and Apache O**

- Points included in Apache 2– Glasgow coma scale, temperature, pulse, respiratory rate, blood pressure, pH, SpO<sub>2</sub>, Hematocrit, TLC, sodium, potassium, creatinine
- Value > 7 suggests severe pancreatitis
- Apache O stands for Apache 2 with O = Obesity with 1 point for BMI = 25–30 kg/m<sup>2</sup> and 2 points for BMI > 30 kg/m<sup>2</sup>.

**D. BISAP (bedside index for severity of acute pancreatitis)**

- Includes blood urea nitrogen, Impaired mental status, SIRS, age and pleural effusion/ascites
- Value > 2 suggest severe pancreatitis.

**E. Baltazar, CT severity index and modified CT severity index***CT severity index*

<b>Pancreatic inflammation</b>	Normal pancreas	<b>0</b>
	Focal or diffuse enlargement of pancreas	<b>1</b>
	Intrinsic pancreatic abnormalities or inflammation of peripancreatic fat	<b>2</b>
	Single ill defined collection or phlegmon	<b>3</b>
	2 or more collections or gas in or adjacent to pancreas	<b>4</b>
<b>Pancreatic necrosis</b>	None	<b>0</b>
	< 30%	<b>2</b>
	30–50%	<b>4</b>
	>50%	<b>6</b>

*Grading of CT severity index*

	<b>Mortality</b>	<b>Morbidity</b>
0–3	3%	8%
4–6	6%	35%
7–10	17%	92%

Baltazar criteria include the same points of first section of CT severity index. That is, it does not take into account the necrosis part of CT severity index and is therefore not used now.

*Modified CT severity index is the latest of the lot and is as follows*

<b>Pancreatic inflammation</b>	Normal pancreas	<b>0</b>
	Intrinsic pancreatic abnormalities and/or inflammation of peripancreatic fat	<b>2</b>
	Pancreatic or peripancreatic fluid collection or fat necrosis	<b>4</b>
<b>Pancreatic necrosis</b>	None	<b>0</b>
	< 30%	<b>2</b>
	>OR = 30%	<b>4</b>
<b>Complications</b>	None	<b>0</b>
	>Or = 1 of Effusion/ ascites/ vascular event/ parenchymal / GIT complication	<b>2</b>

- Grading pattern is similar to CT severity index
- CTSI/MCTSI > 6 is considered severe pancreatitis.

#### **F. ATLANTA criteria**

Organ failure, local complications and presence of systemic complications such as DIC platelet <1,00,000/cu. mm, Fibrinogen < 1g/dl, fibrin split products > 80 microgram/dl and/or calcium < 7.4 mg/dl are considered as manifestations of severe pancreatitis according to Atlanta criteria.

Besides, **modified Marshall organ failure scoring system** and **Goris score** can also be used to assess severity of acute pancreatitis.

#### **Other parameters used include:**

- Hematocrit > 44 %
- C-Reactive protein at 48 hours > 150 mg/dl
- Il-6 > 2.7 pg/ml
- Positive urinary cationic trypsinogen

#### **Q65. Write a note on cystic pancreatic neoplasms.**

**Describe the intraductal papillary mucinous neoplasm of the pancreas.**

**Discuss the features and management of a patient with mucinous cystadenoma of pancreas.**

#### **Ans. A. Mucinous Cystic Neoplasm (Muc tumors)**

- Most common cystic neoplasms of the pancreas
- Can be benign or malignant
- Contain mucin-producing epithelium
- Ovarian-like stroma
- Estrogen and progesterone staining are positive in most cases
- Frequently seen in young women, the mean age at presentation is in the 5th decade
- Typically found in the body and tail of the pancreas.

#### *Diagnoses*

- CECT—Solitary cyst, fine septations, rim of calcification
- Features suggestive of malignancy
  - Larger tumor size
  - The presence of eggshell calcification
  - Mural nodule.
- EUS and cyst fluid analyses—Mucin-rich aspirate high CEA levels (>192 ng/mL) and low levels of cyst fluid amylase.

#### *Management*

- Pancreatic resection
- Although the prognosis of patients who undergo pancreatectomy for invasive MCNs is poor, it is more favorable than that for ductal adenocarcinoma of the pancreas
- Adjuvant chemotherapy is given in node-positive disease.

**B. Serous Cystic Neoplasm (Grand mother tumors)**

- **m.c. site** - Head of the pancreas
- Elderly Females
- Can present as asymptomatic incidental finding or as Vague abdominal pain, weight loss and obstructive jaundice
- SCNs are large, well-circumscribed masses
- Microscopic examination reveals multiloculated, glycogen-rich small cysts
- Central calcification, with radiating septa giving the sunburst appearance, is a radiographic sign on CT
- Pancreatectomy is suggested when the diagnosis of malignancy is uncertain, or in symptomatic serous cystadenomas and in patients with a tumor larger than 4 cm.

**C. Intraductal papillary mucinous neoplasm**

- Also known as mucin-secreting carcinoma, intraductal cystadenoma, villous adenoma of the duct of wirsung, diffuse intraductal papillary adenocarcinoma, mucinous duct ectasia and intraductal papillary mucinous tumor
- Equal incidence in males and females
- Types
  - Main duct type/side branch type/mixed type.
  - Benign/borderline (in-situ)/invasive adenocarcinoma.

**Side branch IPMN**

- Risk of malignant transformation is directly related to the size of the cystic dilation, mural nodules or general thickening of the cyst wall, symptoms such as jaundice, pain, and diabetes
- Asymptomatic lesions smaller than 1 cm - surveillance with CT or MRI in 1 year is appropriate
- Asymptomatic cysts between 1 and 3 cm - Imaging at 6 months is appropriate, followed by annual evaluation if no change in size has occurred
- Cysts larger than 3 cm warrant surgical resection
- Any patient with symptoms or worrisome features related to side branch IPMNs (e.g. jaundice, mural nodule, dilated main pancreatic duct, pain, diabetes) should undergo surgical resection.

**Main Duct Intraductal Papillary Mucinous Neoplasm**

- More chances of malignancy
- Jaundice, elevated serum alkaline phosphatase level, mural nodules, diabetes, and main pancreatic duct diameter of 7 mm or larger are strongly associated with invasive IPMNs
- EUS shows fish mouth sign
- Aspirated fluid is typically viscous and clear and contains mucin. Cytology studies demonstrate mucin-rich fluid with variable cellularity, columnar mucinous cells with variable atypia may also be seen. As in MCNs and side branch IPMNs, fluid aspirates characteristically reveal an elevated CEA level (>192 ng/mL, log scale)
- Surgical resection is indicated in all cases whether symptomatic or asymptomatic.

**Mixed-Type Intraductal Papillary Mucinous Neoplasm** - Surgical resection is indicated for the treatment of mixed-type IPMN.

### **Surgical Resection for Intraductal Papillary Mucinous Neoplasm**

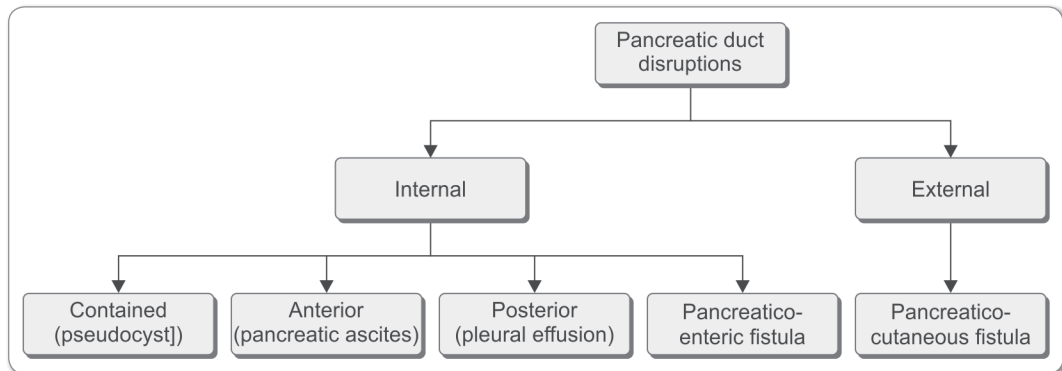
Partial pancreatectomy is the treatment of choice.

**Q66. What is Pancreatic duct disruption? Classify and discuss its management.**

**Discuss the management of a patient with pancreatic ascites.**

**Ans.**

### **Classification**



### **Causes**

- Alcoholic pancreatitis (**most common in adults**)
- Trauma (**most common in children**)
- Gallstones
- Iatrogenic
- Other causes of chronic and acute pancreatitis.

### **Pathogenesis**

Because of all the causes cited above, the final result is acute or chronic pancreatitis which leads to the pancreatic duct disruption which according to the region that it disrupts leads to one of the above mentioned manifestation.

### **Diagnostic criteria**

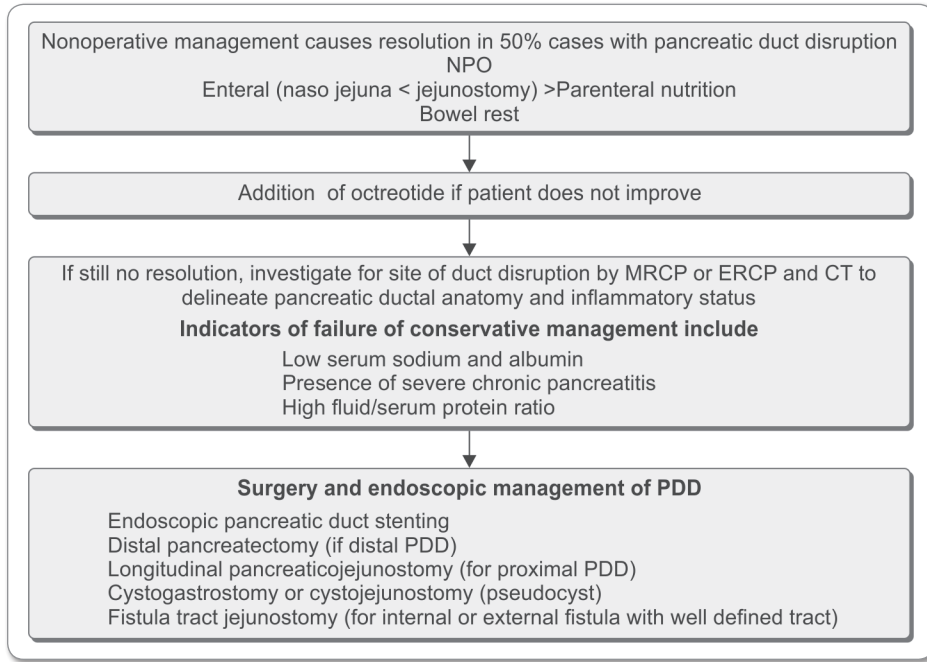
Given by **Cameron et al**

- Fluid amylase > serum amylase
- Fluid albumin > 3 g/dl except in malnourished patients

Serum amylase is also increased in 80 to 90 % patients due to pleural and peritoneal surface absorption of amylase. Fluid is clear/straw colored in most cases.

### **Differential diagnoses**

- Cirrhotic effusion or ascites (albumin < 1.5 g/dl)
- Malignant effusion – tumor markers.

**Management****SPLEEN**

**Q67. Write a shortnote on hypersplenism.**

**Enumerate the causes of massive splenomegaly and discuss hypersplenism.**

**Ans.** Massive splenomegaly is when the spleen is palpable greater than 8 cm below the costal margin or when its drained weight is > or = 1000 gm.

**Causes (Only 10 are there. Memorize them all) are as follows:**

- Chronic myeloid leukemia
- Chronic lymphoid leukemia
- Hairy cell leukemia
- Lymphoma
- Myelofibroses with myeloid metaplasia
- Polycythemia vera
- Gaucher disease
- Sarcoidoses
- Autoimmune hemolytic anemia
- Diffuse splenic hemangiomatosis.



**Hypersplenism**

- It is defined as splenomegaly, cytopenia(s) – anemia, granulocytopenia and/or thrombocytopenia, normal or hyperplastic bone marrow and a response to splenectomy.

**Exception:** Response to splenectomy is not seen to granulocytopenias sometimes and therefore that point in the definition is not absolute.

**Causes of cytopenias**

- Increased destruction of the cellular elements secondary to reduced flow through the enlarged and congested splenic cords or
- Immune mediated mechanisms.

**Types**

Primary (Idiopathic)	Secondary
Cause is the spleen itself without any other known disease affecting spleen	Due to some disease affecting spleen such as rheumatoid arthritis, malignancy (leukemia, lymphoma, metastatic cancer), chronic malaria, tuberculosis, etc.

**Blood picture**

- Anemia, leucopenia, thrombocytopenia can be present
- Bone marrow picture can be seen to rule out the causes of hypersplenism
- Morphology of different cell lines is normal. Though spherocytosis may be seen sometimes
- The reticulocyte index is raised as marrow production of red cells is increased but it may still be less than expected because of the increased splenic destruction.

CT, MRI of abdomen can be used to diagnose the cause of secondary hypersplenism.

**Clinical implications**

- Hypersplenism with thrombocytopenia is common in the patients with cirrheses and is usually the first sign of the development of portal hypertension
- Hypersplenism with rheumatoid arthritis is called **Felty Syndrome**
- Patients can have symptoms and signs related to the underlying disease or the cytopenias
- Complication in unmanaged cases is rupture of spleen which is a dreaded event.

**Management**

- **Secondary hypersplenism** - Therapy for the underlying disease should be attempted prior to consideration of splenectomy
- **Primary hypersplenism** – Splenectomy.

**Indications for Splenectomy in these patients**

- Severe reduction of platelets or immune cells
- Disorders such as leukemia and lymphoma, and metastatic tumors.

**Q68. Discuss management of a patient with splenic injury.****Write a note on spleen injury scale.**

**Ans.** Most common organ injured in blunt trauma abdomen

**The grading of splenic injury is done using CECT abdomen in hemodynamically stable patients**

Organ injury scale is as follows:

Grade	Injury	Extent	Management Stable patient	Management Unstable patient
I	Hematoma Hematoma Laceration	Subcapsular < 10% surface area Parenchymal < 1 cm Capsular tear < 1 cm depth	Conservative	Surgery
II	Hematoma Hematoma Laceration	Subcapsular 10-50% Parenchymal 1-5 cm Capsular tear 1-3 cm depth	Conservative	Surgery
III	Hematoma  Hematoma Laceration	Subcapsular > 50% / expanding/ ruptured/ <b>involve trabecular vessels</b> Parenchymal >5 cm >3 cm depth	Conservative	Surgery
IV	Laceration	Segmental or Hilar vessel injury with > 25% spleen devascularized	Angiography with/out embolisation	Surgery
V	Laceration	Shattered Spleen with/out hilar devascularization	Angiography with/out embolisation	Surgery

**Pathophysiology**

- Direct compression of the organ in the left upper quadrant of the abdomen or
- Deceleration mechanism that tears the splenic capsule or parenchyma, mainly at areas fixed or tethered to the retroperitoneum.

**Management**

- The trend has shifted towards conservative management of patients with splenic trauma when they are hemodynamically stable especially in Grade 1,2 and 3 trauma
- Hemodynamic stability is absence of tachycardia, hypotension, metabolic acidoses and no signs of shock at any time at presentation or after adequate resuscitation
- **In summary**, hemodynamic unstable patients go to the surgery directly.  
In the remaining patients, if the CT shows a grade IV or V injury, or a contrast blush, angiogram is recommended.  
If neither of these is noted, but the hemoglobin continues to decline “too quickly”, or there is a large hemoperitoneum or transient and recurrent hypotension and tachycardia, then also, an angiogram is also warranted in stable patients with any grade of injury.
- **Spleen conserving procedures** are also being recommended for the patients going for surgery such as splenorrhaphy or partial splenectomy depending on the extent of injury (preferred in grade 2/3 patients but not in patients with shattered spleen/complex injuries/multiple organ injuries. Done using monofilament sutures with or without gelfoam or omental patch or by wrapping the spleen in absorbable or nonabsorbable mesh)
- **For other patients**, splenectomy is the treatment of choice

- Vaccination and OPSI prevention should be done as described in the question on OPSI
- Complication of conservative management as well as after angiographic methods is splenic abscess and splenic infarcts. Late pseudoaneurysm is also a common complication
- Most common complication after splenectomy is left lower lobe atelectasis
- Other important complications include OPSI, pneumonia, pancreatic fistula/pancreatitis and wound complications.

**Q69. Enumerate the complications of splenectomy and discuss OPSI.**

**Enumerate the complications and hemodynamic alterations after splenectomy.**

**Ans.** Complications of splenectomy

**Most common complication** – Left lower lobe atelectasis

*Other complications*

<b>Pulmonary</b>	<ul style="list-style-type: none"> <li>• Pneumonia</li> <li>• Pleural effusion</li> </ul>
<b>Pancreatic</b>	<ul style="list-style-type: none"> <li>• Pancreatitis</li> <li>• Pseudocyst pancreas</li> <li>• Pancreatic fistula</li> </ul>
<b>Thromboembolic</b>	<ul style="list-style-type: none"> <li>• Portal vein thrombosis</li> <li>• Deep vein thrombosis</li> </ul>
<b>Local</b>	<ul style="list-style-type: none"> <li>• Wound infection</li> <li>• Subphrenic abscess</li> </ul>
<b>Increased susceptibility to INFECTIONS</b>	<ul style="list-style-type: none"> <li>• <i>S. Pneumoniae</i></li> <li>• <i>H. influenza type B, Meningococcus</i></li> <li>• <i>Salmonella, Bartonella, Bacteroids, Enterococcus</i></li> <li>• <i>Capnocytophaga canimorsus</i></li> <li>• <i>Plasmodium, Babesia, Erhlichia (PROTOZOA)</i></li> </ul>

**Hematologic consequences of splenectomy**

<b>Immediate (normalize within 2-3 weeks)</b>	<ul style="list-style-type: none"> <li>• Leucocytosis</li> <li>• Thrombocytosis</li> </ul>
<b>Chronic consequences</b>	<ul style="list-style-type: none"> <li>• Howell-Jolly bodies (nuclear remnants)</li> <li>• Heinz bodies (denatures hemoglobin)</li> <li>• Pappenheimer bodies (iron globules)</li> <li>• Anisocytosis and poikilocytosis</li> <li>• Basophilic stippling and nucleated erythrocytosis</li> </ul>

**Overwhelming post splenectomy infection**

- **Incidence** – 1 to 5%
- Most common fatal late complication of splenectomy.

**Duration**

- Most infections occur more than 2 years after splenectomy and 42% occur more than 5 years after splenectomy.

**Organisms**

- *S. pneumoniae* (m.c., 50% and 90% of cases)
- Other organisms - *H. influenzae, N. meningitidis, Streptococcus* and *Salmonella* spp., other pneumococcal organisms, and *Capnocytophaga canimorsus* (dog bites).

**Pathogenesis**

- The inability of spleen to filter and phagocytose bacteria and parasitized blood cells predispose a person to infections especially with encapsulated bacteria. Also the antibody production function of spleen is lost
- There is decrease in the amount of tuftsin and properdin in the body and therefore decreased opsonization function
- The body responds to the old and known antigens in the same way after splenectomy as it used to be before. But, the response to the new antigens is diminished.

**Risk factors**

- When splenectomy done for malignancy or hematologic conditions than for those who underwent splenectomy for trauma
- Young children (<4 years of age).

**Clinical features**

- **Prodromal phase**—Fever, rigors, and chills and other nonspecific symptoms including sore throat, malaise, myalgias, diarrhea, and vomiting.
- Pneumonia, meningitis, high-grade primary bacteremia can occur.
- **Late phase**—Hypotension, disseminated intravascular coagulation, respiratory distress, coma, and death within hours of presentation.
- Mortality rate is between 50% and 70% for florid OPSI.
- Sequelae - Peripheral gangrene requiring amputation, deafness from meningitis, mastoid osteomyelitis, bacterial endocarditis, and cardiac valvular destruction.

**Preventive measures**

- Appropriate and timely immunization
- Education and
- Prompt treatment of infection
- The benefit of prophylactic antibiotics in this setting remains controversial
- Vaccination against *Pneumococcus*, *Meningococcus* (both repeated every 5 years) and *H. influenzae* (repeated every 10 years). Yearly influenza vaccination has also been recommended.

These vaccines should be given about 2 weeks before elective surgery and as soon as possible after emergency surgery.

## HERNIAS

**Q70. Discuss the types of meshes used in hernia surgery.**

**Why are meshes used in hernia surgery? Enumerate the various types.**

**Ans.**

**Primary repair**

Done in defect <3 cm in diameter with viable surrounding tissue in a hernia which is clearly a result of technical error at the initial operation such as suture fracturing.

**Mesh repair**

For all hernias with defect >3 cm, mesh repair is preferred because primary repair has a recurrence rate of 10 to 50% which becomes 5 to 25% with mesh placement.

**Positions of mesh placement in open surgery**

<b>Overlay</b>		Just below subcutaneous tissue
<b>Interposition</b>		Along the edge of the defect
<b>Intermuscular</b>		Between muscular layers of the anterior abdominal wall
<b>Inlay</b>	<b>Retrorectus</b>	Rives stoppa wantz repair- mesh placed between rectus muscle and posterior rectus sheath
	<b>Preperitoneal</b>	Between posterior rectus sheath and peritoneum
	<b>Intraperitoneal (underlay)</b>	Also called sublay

**Position of placement of mesh in laparoscopy can be**

- Intraperitoneal onlay (IPOM)
- Preperitoneal (space of Bogros).

**Ideal mesh**

- Chemically inert
- Resistant to mechanical stress and at the same time compliant
- Sterilizable
- Noncarcinogenic
- Minimal inflammatory reaction
- Hypoallergic.

**Types of meshes**

<b>Weight</b> (depends on pore size and type of filament)	<ul style="list-style-type: none"> <li>• Light weight</li> <li>• Heavy weight</li> </ul>	<50g/m <sup>2</sup> >80g/m <sup>2</sup>	Increased flexibility and decreased discomfort. Superior to heavy weight. Pain, heaviness are more.
<b>Pore size</b>	<ul style="list-style-type: none"> <li>• Micro</li> <li>• Macro</li> </ul>	80-800 micrometer 1-3mm	Light weight macroporous are considered the best meshes as they have increased elasticity, flexibility and less chances of shrinkage, less pain and less scar plate formation
<b>Reaction to water</b>	Hydrophobic Hydrophilic		

**Synthetic meshes**

<b>Polypropylene</b>	<ul style="list-style-type: none"> <li>• Extraperitoneal placement</li> <li>• Hydrophobic, macroporous, light weight</li> </ul>
<b>Polyester</b>	<ul style="list-style-type: none"> <li>• Extraperitoneal placement</li> <li>• Hydrophilic, heavy weight, macroporous, multifilament</li> </ul>
<b>Single sheet mesh (ePTFE)</b>	<ul style="list-style-type: none"> <li>• Intraperitoneal placement</li> <li>• Visceral side is microporous and abdominal wall side is macroporous</li> <li>• Hydrophobic</li> <li>• Not incorporated into native tissue, only encapsulated and therefore has high rate of infection</li> </ul>

Contd...

Contd...

<b>Composite mesh</b>	<ul style="list-style-type: none"> <li>One material is on visceral side (ePTFE) and other on parietal side (polypropylene)</li> </ul>
<b>Barrier mesh</b>	<ul style="list-style-type: none"> <li>Absorbable anti adhesive barrier of oxidized regenerated cellulose/ omega three fatty acids/ collagen hydrogels over visceral side of polypropylene or polyester meshes. Not validated yet.</li> </ul>

**Biologic meshes**

- Basically composed of extracellular matrix collagen stripped off all of its cellular elements
- Source can be human dermis (requires processing before application), bovine pericardium or porcine dermis/small intestinal submucosa
- Used in infected/contaminated areas where synthetic meshes are relatively contraindicated
- Inlay is the most preferred approach followed by onlay but never in interposition
- They provide matrix for neovascularization and native collagen deposition.

**Q71. Write a note on management of incisional hernia.**

**Enumerate the factors for development of an incisional hernia and discuss its management.**

**Ans. Factors for development of an incisional hernia**

Patient factors	Operative factors
<b>Old age, male gender</b> <b>Malnutrition in perioperative period</b> Severe anemia, hypoproteinemia, vitamin A,C deficiency, zinc deficiency, advanced malignancy related cachexia, patients of longstanding dysphagia/gastric outlet obstruction. <b>Obesity and sleep apnea</b> <b>Diabetes mellitus</b> <b>Chronic steroid use</b> <b>Straining factor</b> Chronic constipation, chronic cough, Bladder outlet obstruction, heavy weight lifting, ascites, prolonged ileus	<b>Type of operation</b> <ul style="list-style-type: none"> <li>Emergency surgeries for peritonitis, cancer, pancreatic surgeries, appendicular abscesses</li> </ul> <b>Incisions</b> <ul style="list-style-type: none"> <li>Midline infraumbilical incision, Kocher's incision, McBurney's incision</li> </ul> <b>Technique</b> <ul style="list-style-type: none"> <li>Mass closure</li> <li>Nonanatomical closure</li> <li>Suturing under tension</li> <li>Wound infection or hematoma</li> <li>Closure with absorbable sutures</li> <li>Continuous suturing</li> <li>Drains through the main wound</li> </ul>

**Nonoperative management**

For patients who are not medically fit or who deny surgery, use of abdominal binders is a palliative approach to prevent development of complications in the hernia.

**Operative management**

**Procedure of choice** is laparoscopic intraperitoneal onlay (IPOM) mesh hernioplasty.

**Other options include the following**

- Repair by **Mayo's technique** of double breasting (described in umbilical hernia repair)
- Component separation technique** (Ramirez open technique or endoscopic component separation)

- The principle here is to shift the external oblique aponeurosis and rectus sheath medially so as to cover the defect in midline. For this, release incisions are made laterally and the structures are brought near midline to bridge the gap and then they are sutured together
- The relaxing incision is made 2 cm lateral to the linea semilunaris on the lateral external oblique aponeurosis from several centimeters above the costal margin to the pubis
- The external oblique is then bluntly separated in the avascular plane, away from the internal oblique, allowing its medial advancement
- Further relaxing incisions can be given to aponeurotic layers of the internal oblique or transversus abdominis but this can result in problematic lateral bulges or herniation at the site of relaxing incision. Additional release can be safely achieved by incising the posterior rectus sheath
- This can give nearly 20 cm closure margin. However, a lateral bulge can occur after releasing the external oblique aponeurosis

**Endoscopic component separation:** The basic principle of a minimally invasive component separation is to gain direct access to the lateral abdominal wall without creating a lipocutaneous flap

- Typically, this is performed by a direct cut down through a 1-cm incision off the tip of the 11th rib overlying the external oblique muscle. The external oblique is split in the line of its fibers and a standard bilateral inguinal hernia balloon dissector is placed in between the external and internal oblique muscles, toward the pubis and then the same procedure is carried out endoscopically.
- Open inlay mesh repair (write from the SN on meshes used in hernia repair)
- Lattice or darning  
Defect is closed with the fascial sutures in an interlacing manner between the muscle and aponeurosis of either side
- Repair in layers- Cattle's repair  
Defect repair is done in multiple layers –
  - Neck of sac
  - Cut edges of base of sac
  - Fascia around the peritoneal sac
  - Muscles are apposed over this
  - Anterior rectus sheath
  - Skin
- Keel operation

This is the procedure where the hernia sac is not opened but, inverted and kept in that position by a series of inverting or pleating sutures (3-4 layers). Lastly, anterior sheath and skin are closed.

### Results

Without mesh the recurrence rates are 30 to 40%, which decreases to 10% with the use of mesh and therefore mesh repair is the favored treatment approach over anatomical repair.

**Q72. What is umbilicus? Explain the anatomy of Umbilicus.****Enumerate the contents of umbilical ring and discuss its anatomy.**

**Ans.** Embryologically, the umbilicus is a midline fusion of the medial aponeurotic borders of both rectus abdominis and aponeuroses around the umbilical cord. This fusion may take place around the 10th week, after the herniated midgut returns to the peritoneal cavity. During the period of fetal circulation, the following embryologic entities are found at the umbilicus:

- Left umbilical vein
- Vitellointestinal duct
- Vitelline artery and vein
- Urachus
- Two umbilical arteries

**Structures Associated with the Umbilical Cord and Umbilicus**

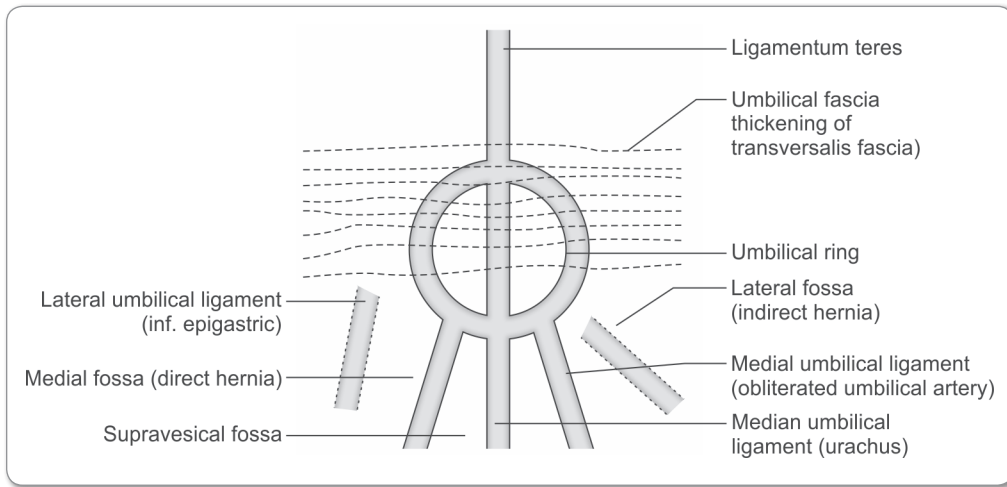
In the Primitive Body Stalk	In the Neonatal Abdomen
Yolk stalk (vitelline duct)	Absent
Extraembryonic coelom	None
Herniated intestine	Returned to abdomen
Vitelline arteries	Celiac, superior, and inferior mesenteric arteries
Vitelline veins	Part of portal vein
Allantois	Urachus (median umbilical ligament)
Umbilical arteries	Medial umbilical ligaments
Umbilical veins	Round ligament in falciform ligament
Undifferentiated mesenchyme	None

- Remember the following four anatomic entities, which pass through the umbilical ring in the **newly born child**:

Left umbilical vein (round ligament of the liver), Urachus (median umbilical ligament) and two umbilical arteries (medial umbilical ligaments)

- The umbilicus is located at the center of the umbilical region. For all practical purposes, the umbilicus is a scar. It is not the same in all individuals. Its boundaries are the epigastric area above, the hypogastric area below, and the right and left lumbar areas laterally
- The umbilicus is essentially at the vertical midpoint of the linea alba. It marks the junction of the lower end of the well-formed upper linea alba and the beginning of the poorly-defined lower linea alba
- The medial umbilical ligaments (obliterated umbilical arteries) and the urachus (obliterated allantoic duct) participate in the formation of the fibrous umbilical ring. The round ligament (obliterated umbilical vein) arises from the inferior margin of the ring and passes superiorly in the falciform ligament.
- The umbilical ring and its four essentially solid tubes (two obliterated umbilical arteries, the urachus, and the round ligament) are related as follows:





**Fig. 13:** Umbilical ring anatomy

### **Umbilical ligaments**

- In 74% of cases, the ligamentum teres of the liver crosses the umbilical ring, and attaches to its lower margin
- In 24%, the ligamentum teres splits and attaches to the upper margin of the ring, forming a triangle. The urachus also splits to form another triangle related to the lower margin of the ring. It is possible that the structure and manner of formation of these triangles are involved in the genesis of supraumbilical or infraumbilical hernias.

### **Umbilical fascia**

- In 36%, a localized thickening of the transversalis fascia in this area, named the umbilical fascia, covers the umbilical ring in toto. This fascial "buffer" can protect against the genesis of an umbilical hernia
- In 38% of individuals, the umbilical fascia covers only the upper part of the umbilical ring
- In 6% of individuals, the umbilical fascia covers only the lower part of the umbilical ring
- In 4% of individuals, the umbilical fascia is located above the ring
- In 16% of individuals, the umbilical fascia is absent.

### **Q73. Write a note on Umbilical hernia.**

#### **Discuss omphalocele in brief.**

#### **What are the types of umbilical hernia? Discuss their management in brief.**

#### **Ans. Pediatric umbilical hernia**

Congenital anterior abdominal wall defects are as follows:

- Defect <4 cm in the region of umbilical cord – hernia of umbilical cord
- Defect > 4 cm at this site – omphalocele (exomphalos)
- Defect of around 4 cm to one side (commonly right) of the umbilical cord – gastroschises.

#### *Exomphalos*

- Hernia occurring through the umbilical ring

- Occurs due to failure of migration and fusion of lateral folds of the anterior abdominal wall
- Has a transparent sac formed of peritoneum and amniotic membrane with Wharton's jelly in between
- Content may be all the midgut or part of it
- **Exomphalos is called minor variety when <5 cm and major variety when > 5 cm**
- Associated diseases with this anatomical disorder are Beckwith Wiedemann syndrome, Trisomy 13, 15, 18, 21 and Cantrell's pentalogy.
- **Management**

Immediate surgical repair is the treatment procedure of choice.

– *Preoperative management*

- Keep patient nil per oral and start IV fluids
- Nasogastric decompression
- Avoid hypothermia
- Start IV antibiotics
- Identify associated anomalies
- The sac is protected with wet saline mops or antiseptic sterile dressing before surgery.
- Don't excise the omphalocele sac as unique treatment with escharotics agents can be done such as betadine ointment/ silver nitrate/mercurochrome which helps to shrink the sac which can be repaired at a later date

– *Surgical options*

- Primary closure
- Staged skin flap closure
- Staged silastic pouch closure.

Whichever technique is used, cardiopulmonary compromise must be avoided in the patient.

*Gastroschisis*

- There is a full thickness defect in the anterior abdominal wall on the right side of the umbilical cord
- This is a vascular event where the hernia occurs because of a defect at the site of umbilical vein
- Associated anomalies include intestinal atresia and undescended testis
- The survival rates in gastroschisis are better than in omphalocele but, chances of obstruction are more
- It is more common in the children of Young mothers (< 20 years), maternal smoking, alcohol, recreational drug use, maternal use of aspirin, ibuprofen, pseudoephedrine in the first trimester
- It requires immediate surgical management
- Good prognoses than omphalocele.

*Surgical options*

- Primary repair
- Biological mesh repair/prosthetic mesh repair
- Silo placement and staged surgery
- Fluid requirements are more in people with gastroschisis and therefore 1.5 times the normal requirement is administered

- Treatment for the associated intestinal atresia is done at 6 to 8 weeks
- It has 20% incidence of necrotizing enterocolitis
- There is also a high chance of prolonged postoperative ileus.

**Adult umbilical hernia (Paraumbilical hernia)**

It occurs through a defect in the linea alba

*Risk factors*

- Women are more commonly affected, especially multiparous
- Obesity
- Straining is a predisposing factor – chronic constipation/chronic cough/urinary obstruction.

*Contents of sac* – portions of small intestine, large intestine, greater omentum.

*Clinical features include:*

- Presence of a swelling in the umbilical region with one of the edges of umbilicus involved and increase in size on straining
- Patient also experiences pain in the swelling, intermittent attacks of intestinal colic or intestinal obstruction
- Swelling is firm, round, and has a positive cough impulse with partial reduction possible. The hernia is usually partially reducible due to presence of adhesions between contents and the sac
- On palpation, the defect in the linea alba can be felt after reducing the sac.

*Complications:*

- Irreducibility
- Obstruction
- Incarceration
- Strangulation.

*Management*

Treatment is always surgery

**• Preoperative measures**

- Correction of straining factors such as weight reduction, urinary tract management, treatment of chronic cough and constipation
- Stop cigarette smoking for atleast a month if not more or permanent.
- Consent for umbilicectomy is important.

**Anatomical repair is by Mayo's operation important steps of which are as follows:**

- Transverse incision encircling the umbilicus is given such that it extends beyond the swelling laterally.
- It is deepened to expose the rectus sheath anterior layer
- Mobilization of upper and lower skin flaps is done to about 5 cm beyond the rim of the gap
- Than, neck of the sac is opened and contents reduced
- Non absorbable vertical mattress sutures are used to double breast the edges of rectus sheath with peritoneum such that the lower flap is placed underneath the upper flap
- Abdominal wall is closed in layers above this.

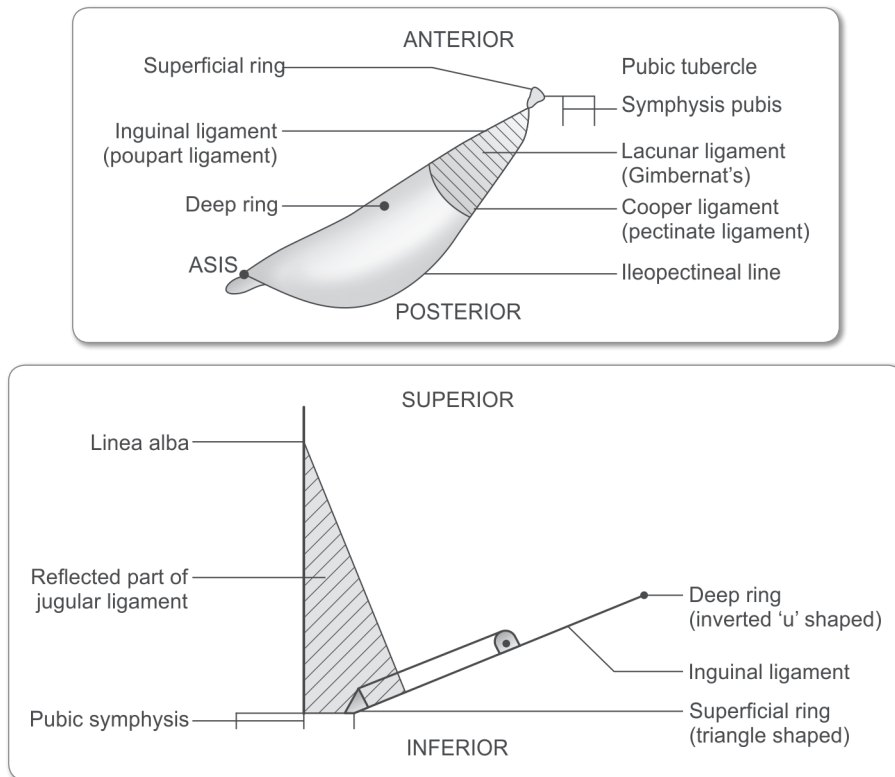
**Other option is the mesh repair after umbilicectomy.**

• **Postoperative measures**

- Gastric suction and intravenous fluids, nutrition and vitamins till patient passes flatus.
- Avoid straining factors.
- Adequate antibiotic therapy
- No strenuous work for at least 3 months and use of abdominal binder for at least 3 months
- Avoid weight gain and obesity.

**Q74. Explain the open and laparoscopic anatomy of inguinal region.**

**Ans.**



**Fig. 14:** Inguinal canal open anatomy

**Open anatomy of Inguinal canal**

- It is an oblique canal, approximately 4 cm long extending from deep inguinal ring to superficial inguinal ring.
- It is located at the inferior margin of the anterior abdominal wall, parallel and superior to the medial half of the inguinal ligament.

**Openings**

*Deep inguinal ring:*

- Internal entrance to the canal through the defect in the transversalis fascia

- It is located 1.25 cm superior to the midpoint of the inguinal ligament, lateral to the inferior epigastric vessels
- It is **inverted U shaped** and the arms of the U are formed by thickenings of transversalis fascia whereas the inferior part is formed by Iliopubic tract (another thickening of transversalis fascia).

#### *Superficial ring:*

- External exit through the **external oblique aponeurosis**, located superolateral to the pubic tubercle
- It is a **triangular opening** whose base is formed by pubic tubercle and the two other sides (superomedial) and (inferolateral) are formed by the external oblique aponeurosis and inguinal ligament respectively.

#### *Boundries*

- **Anterior wall:** External oblique aponeurosis, and internal oblique laterally
- **Posterior wall:** Transversalis fascia laterally, Internal oblique and conjoint tendon (joint insertion of aponeurosis of internal oblique and transverses abdominus) medially
- **Roof:** Arched fibers of internal oblique
- **Floor:** Inguinal ligament, reinforced medially by the lacunar ligament

#### **Contents of Inguinal Canal**

- Spermatic cord in men
- Round ligament in women
- Ilioinguinal nerve
- Blood and lymphatic vessels.

#### **Contents of Spermatic Cord**

- Vas deferens
- Remnant of processus vaginalis

#### *Arteries*

- Artery to vas
- Testicular deferens artery
- Cremasteric artery

#### *Nerves*

- Genital branch of genitofemoral nerve
- Sympathetic plexus around the artery to vas deferens.

#### *Veins*

- Pampiniform plexus and testicular veins
- Cremasteric veins
- Deferential veins.

#### **Lymphatics of these structures**

#### **Coverings of Spermatic Cord**

- Internal spermatic fascia from fascia transversalis
- Cremasteric fascia from internal oblique and transverses abdominis aponeurosis
- External spermatic fascia from external oblique aponeurosis.

**Defence Mechanism of Inguinal Canal**

- Obliquity of inguinal canal
- Arching of conjoint tendon
- Shutter mechanism of internal oblique
- Ball valve mechanism due to contraction of cremaster muscle which plugs to superficial ring
- Slit valve mechanism: When external oblique muscle contracts, intercrural fibers of superficial ring appose.

**Boundaries of the Hesselbach triangle (The important triangle in open anatomy)**

- **Medial:** Lateral border of rectus abdominis
- **Lateral:** Inferior epigastric vessels
- **Inferior:** Inguinal ligament

**Hernias within this triangle are direct hernias**

**Hernias lateral to this triangle are indirect hernias**

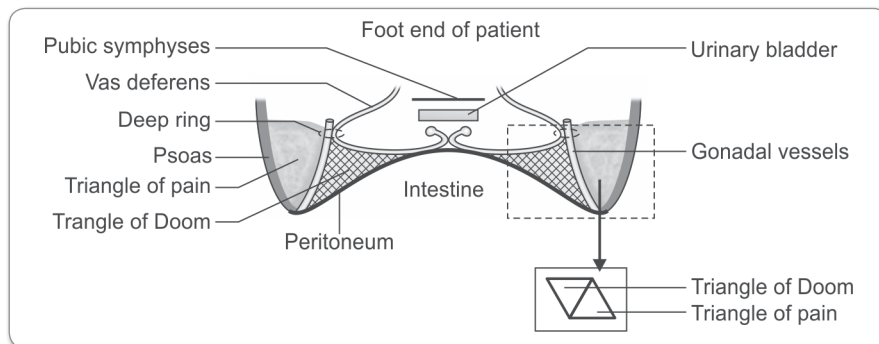
**Laparoscopic anatomy**

- The laparoscopic anatomy means the region is seen from within the abdomen as seen in the laparoscopic total extraperitoneal hernia (TEP) or transabdominal preperitoneal (TAPP) hernia surgery – basically the anatomy as seen in the extraperitoneal/preperitoneal space of Bogros.

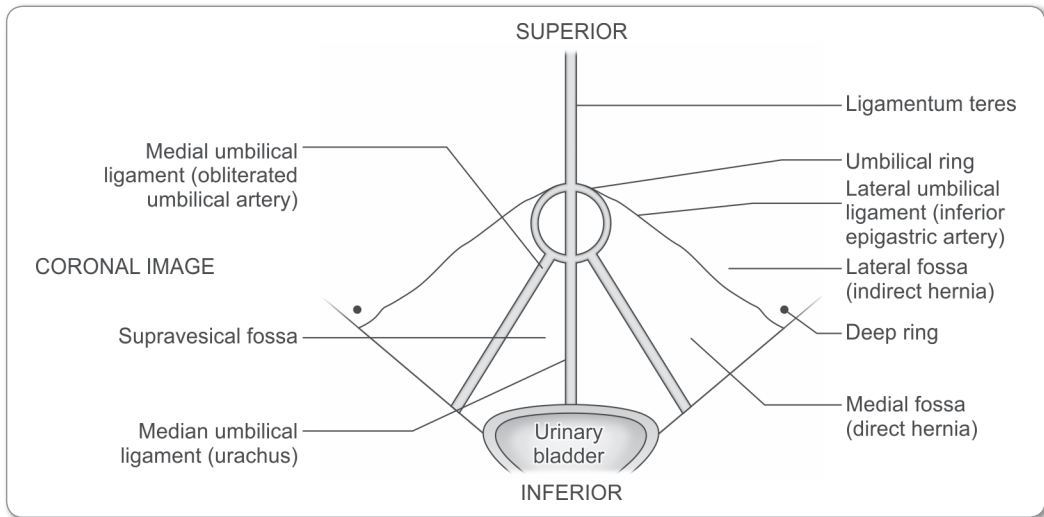
**(Always remember:** In **TEP**, the extraperitoneal space is entered directly by using the open Hasson's technique to place the first umbilical port in the plane between the rectus muscle and posterior rectus sheath and progress downwards. As we go inferiorly, the posterior rectus sheath is defective at the level of arcuate line and so we enter the space between fascia transversalis and peritoneum.

In **TAPP**, the initial access is same as in laparoscopic cholecystectomy, that is through closed technique of port placement into the peritoneal cavity and then the peritoneum is incised in inguinal region to gain access into the space of Bogros i.e. the extraperitoneal space from this window.)

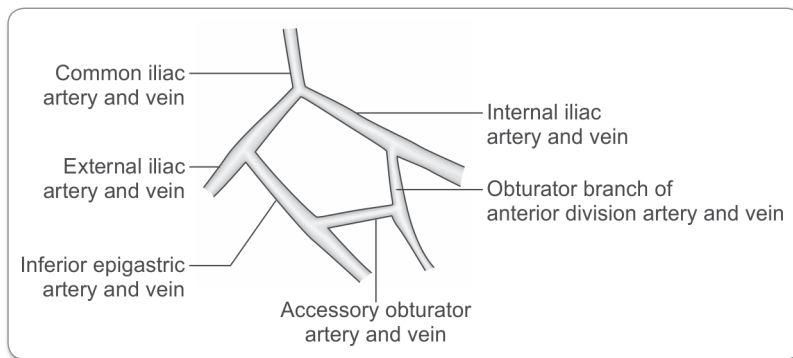
The anatomy is described using three figures which are as follows:



**Fig. 15.1:** Shows the landmarks as seen when the laparoscope enters the space of Bogros, and the two important triangles: triangle of Doom and triangle of pain



**Fig. 15.2:** Shows the important fossae and the structures forming them. These are the potential spaces of hernia formation



**Fig. 15.3:** Shows the formation of corona mortis as it does beneath the triangle of Doom

**Boundaries of the myopectineal orifice of Fruchaud** (The area where all the hernias occur and therefore the area that is to be covered in laparoscopic or open hernia surgery to attempt total treatment of all hernias)

- **Superior:** Arch of internal oblique muscle and transversus abdominis muscle
- **Lateral:** Iliopsoas muscle
- **Medial:** Lateral border of rectus muscle and anterior lamina of rectus sheath
- **Inferior:** Pecten pubis with the Iliopectineal ligament
- **Posterior surface:** Formed entirely by fascia transversalis. The inguinal ligament divides this framework.

The area is traversed by the spermatic cord and femoral vessels.

**Boundaries of triangle of Doom**

- **Medial—**Vas Deferns
- **Lateral—**Gonadal vessels

- **Base** is towards head end of patient and formed by the reflection of the peritoneum. Corona mortis lies beneath it and therefore it is called the triangle of doom

**Vessels forming the corona mortis are:**

- External iliac artery
- Inferior epigastric artery
- Anterior division of internal iliac artery
- Obturator artery
- Accessory obturator branch of Inferior epigastric artery.

**Q75. Write differences between direct and indirect hernia.**

**Ans.**

(**Always remember:** The best way to differentiate direct and indirect hernia is to open and see which it is. All other tests are speculations and not confirmatory.)

Direct hernia	Indirect hernia
Less common (35%)	More common (65%)
<b>History</b>	
<ul style="list-style-type: none"> <li>• More common in elderly</li> <li>• Women almost never have direct hernia</li> <li>• More commonly bilateral</li> <li>• Small in size and not usually reach the base of the scrotum</li> <li>• Reduces easily on lying down or by patient</li> <li>• History of irreducibility is rare</li> </ul>	<ul style="list-style-type: none"> <li>• More common in young</li> <li>• Both sexes can be affected, but, more in males</li> <li>• Commonly unilateral</li> <li>• May be large and can reach the base of the scrotum</li> <li>• May be difficult to reduce</li> <li>• History of irreducibility is possible</li> </ul>
<b>Examination</b>	
<ul style="list-style-type: none"> <li>• Globular in shape with wide neck</li> <li>• Not reach the root of scrotum</li> <li>• Easily pops out on coughing, straining or standing</li> <li>• Easily reduces on lying down</li> <li>• Located medial to the inferior epigastric vessels i.e. within the Hasselbach's triangle.</li> </ul>	<ul style="list-style-type: none"> <li>• Pyriform/ oval in shape with narrow neck</li> <li>• May reach the root of the scrotum</li> <li>• Slowly appears on coughing, straining or standing</li> <li>• Slowly reduces on lying down or manipulation</li> <li>• Located lateral to inferior epigastric vessels i.e. lateral to the Hasselbach's triangle</li> </ul>
<b>Tests</b>	
<ul style="list-style-type: none"> <li>• <b>On Deep ring occlusion</b>, the swelling appears medial to deep ring</li> <li>• <b>On invagination test</b>, the swelling is felt at the pulp of the finger</li> <li>• <b>On Zieman test</b>, the cough impulse is on the middle finger</li> </ul>	<ul style="list-style-type: none"> <li>• <b>On Deep ring occlusion</b>, the swelling does not appear till the ring is occluded</li> <li>• <b>On invagination test</b>, the swelling is felt at the tip of the finger</li> <li>• <b>On Zieman test</b>, the cough impulse is on the index finger</li> </ul>
<b>Complications</b>	
Irreducibility, obstruction, strangulation can occur but are very rare due to wide neck	Irreducibility, obstruction, strangulation can occur and incidence increases as the time progresses

Contd...



Contd...

Direct hernia	Indirect hernia
<b>Coverings</b>	
<ul style="list-style-type: none"> <li>• Peritoneum</li> <li>• Extraperitoneal tissue</li> <li>• Fascia transversalis</li> <li>• Conjoint tendon (Internal oblique and transverses abdominis aponeurosis)</li> <li>• External oblique aponeurosis</li> <li>• Superficial fascia [Scarpa (deep) and Camper (superficial)]</li> <li>• Skin</li> </ul>	<ul style="list-style-type: none"> <li>• Peritoneum sac</li> <li>• Extraperitoneal tissue</li> <li>• Internal spermatic fascia (from Fascia transversalis)</li> <li>• Cremasteric fascia (from Internal oblique)</li> <li>• External Spermatic Fascia (from External oblique)</li> <li>• Superficial fascia [Scarpa (deep) and Camper (superficial)]</li> <li>• Skin</li> </ul>

**Q76. Enumerate the types and classifications of inguinal hernia.**

Ans.

- **Direct and Indirect hernia (Discussed below)**
- **Based on content :** Enterocoele (intestine), omentocoele (omentum), Retroperitoneal organs (**sliding hernia**), part of circumference of bowel (**Richter hernia**), Meckel's diverticulum (**Littre's hernia**), Two intestinal loops with the common part in abdomen (**Maydl's W shaped**), ovary, appendix (**Amyand's Hernia**)
- **Based on extent: Bubonocoele** (hernia ends proximal to superficial inguinal ring)
- **Funicular** (sac of hernia is separate from the sac of testes), **Vaginal** (Hernia is complete and incorporates testes due to a patent processus vaginalis)
- **Congenital v/s acquired hernia** – Congenital occurs because of patent processus vaginalis. Presents as bubonocoele, funicular or vaginal hernia.

**Risk factors of acquired hernia are as follows:**

<b>Congenital</b> Marfan syndrome, Ehlers-Danlos syndrome, Osteogenesis imperfect Patent processus vaginalis/ Canal of Nuck Bladder exstrophy and Prune belly syndrome	<b>Iatrogenic</b> Previous surgery – McBurney's incision for appendicitis causes damage to iliohypogastric nerve and predisposes to hernia
<b>Increased intra-abdominal pressure</b> Obesity, Ascites, pelvic tumors Chronic cough, constipation, bladder outlet obstruction, pregnancy Heavy weight lifting Chronic ambulatory peritoneal dialysis patients	<b>Other acquired factors</b> Smoking Chronic cachexia/malnutrition Advancing age

- **Nyhus Classification**

<b>Type I</b>	Indirect, internal ring normal- patent processus vaginalis
<b>Type II</b>	Indirect, internal ring enlarged, posterior wall of canal normal
<b>Type III A</b> <b>Type III B</b> <b>Type III C</b>	Direct Indirect, posterior wall damaged, sliding/ direct with indirect [Pantaloon] Femoral

Contd...

Contd...

Type IV A	Recurrent direct
Type IV B	Recurrent indirect
Type IV C	Recurrent femoral
Type IV D	Recurrent pantaloons

- **Gilbert Classification**

Type I	Indirect, small
Type II	Indirect, medium
Type III	Indirect, large
Type IV	Direct large, entire posterior wall damaged
Type V	Direct, diverticular type
Type VI	Pantaloons
Type VII	Femoral

- **Complete (reaches the base of scrotum) and Incomplete**
- **Uncomplicated (reducible) and complicated**

*Complications include:*

- Irreducibility
- Obstruction (is usually painless, lax, nontender) – Irreducibility with obstruction of the lumen of content
- Strangulation (Painful, tense, tender) - Irreducibility with arrest of blood supply and obstruction
- Incarceration – content of sac is fixed in sac due to adhesions or size and gets indented like putty with finger tip because of fecal content. This is considered a variety of irreducible hernia
- Inflammation of the contents of hernia such as appendicitis, Meckel's diverticulitis, tuberculosis of the contents, omental infarction
- Hydrocele of the hernia sac – Gibbon's hernia.

**Q77. Enumerate the options in treatment of a patient with inguinal hernia.**

**Ans. Nonoperative management (Truss, Taxis)-** used only if surgery is not possible because of comorbidities.

**Operative management**

- **Herniotomy:** Excision of sac only. Done for congenital hernia and congenital hydrocele.
- **Herniorrhaphy:** Herniotomy followed by reconstruction of the posterior wall of the inguinal canal with body tissues in that area. For examples – Bassini repair (suture conjoined tendon to intumed edge of the inguinal ligament), Shouldice repair, McVay's repair (Cooper ligament), Lytle's repair (repair of the internal inguinal ring), Marcy's repair and so on.
- **Hernioplasty:** Herniotomy with reconstruction of the posterior wall with a prosthetic material.

The aim of using the prosthetic material is to decrease the tension due to forced approximation in herniorrhaphy procedures and thereby decrease recurrence. It can be done by open or laparoscopic technique.

- **Open:** Lichtenstein tension free hernia repair, Gilbert patch and plug repair, Rives stoppa preperitoneal mesh repair technique, Kugel's technique.
- **Laparoscopic:** Total extraperitoneal repair (TEP) and transabdominal preperitoneal repair (TAPP)

Open	Laparoscopy
<ul style="list-style-type: none"> <li>• Inguinal approach cannot cover the entire myopectineal orifice</li> <li>• Bilateral procedure will require separate incisions</li> <li>• Is difficult in recurrent hernias as a virgin plane is difficult to achieve from anterior approach</li> <li>• Has a higher incidence of wound problems</li> </ul>	<ul style="list-style-type: none"> <li>• Entire myopectineal orifice can be easily covered</li> <li>• Bilateral procedure can be done using the same port placement and therefore avoids more incisions</li> <li>• Is the procedure of choice in recurrent hernias</li> </ul>
<ul style="list-style-type: none"> <li>• Immediate recovery takes time</li> <li>• Is less costly</li> <li>• Has a shorter learning curve</li> <li>• Can be done under local anesthesia</li> <li>• Widespread availability of surgeons to teach as well as offer this procedure to the patients.</li> </ul>	<ul style="list-style-type: none"> <li>• Smaller incision therefore less wound infections, hematomas, less morbidity in immediate postoperative period. However, the 6 month incidence of chronic inguinodynia is same to open procedure</li> <li>• Early return to work</li> <li>• Is expensive</li> <li>• Has a definite learning curve</li> <li>• Requires general anesthesia and therefore not possible in patients with comorbidities precluding general anesthesia.</li> </ul>

However, management of a case of strangulated inguinal hernia is different in that the early phase totally depends on resuscitation.

### Diagnoses is always clinical.

#### Step 1 – Resuscitation

- Fluid balance, electrolyte management, correction of acid- base balance once the fluid balance is taken care of are the life saving measures before surgery
- Nasogastric tube placement for gastrointestinal decompression
- Monitoring of urine output, Intake of fluids, pulse, blood pressure, temperature and respiratory rate is very important during this phase of resuscitation
- If necessary, a central venous line can be inserted to give fluid as per central venous pressure measurement
- Antibiotics are to be given.

#### Step 2

- Once the patient is resuscitated, take him to surgery as soon as possible
- It should never be reduced before surgery
- Hernia can be explored from the groin approach as in elective cases
- Difference from elective cases is that the sac is to be opened from fundus, all the toxic fluid drained and bowel assessed for viability. Once all these steps are done, only then

the neck is opened. The basis for this step is that if the sac is opened at the neck first, all the toxic fluid will contaminate the peritoneal cavity and if there is a gangrenous segment, it can also fall back into the abdomen and may necessitate a midline laparotomy. As such, if resection and anastomosis is required, that in itself may require a laparotomy to reposit the anastomosed segment back or to get adequate length of bowel for anastomosis

- Other difference is that the normal prosthetic mesh cannot be placed in the infected area and therefore it requires a biological mesh or an anatomical closure (herniorrhaphy). Also, the patient will require postoperative antibiotics if there was gangrene which is not the case after elective surgery.

#### Q78. Write a note on femoral hernia.

Ans.

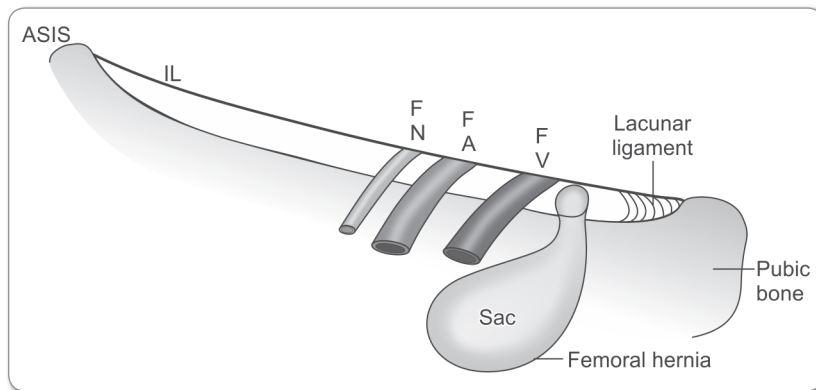


Fig. 16: Anatomy of the femoral canal

#### Anatomy of femoral canal

- Most medial compartments of femoral sheath extending from femoral ring above to saphenous opening below.
- It is similar to truncated cone which is narrow at the femoral ring.
- It is 1.25 cm long and 1.25 cm wide at its base, which is directed upwards.

#### Femoral sheath

- Its anterior layer is formed by fascia transversalis and posterior layer formed by fascia iliaca
- Femoral canal is the medial most compartment. Femoral vein in the middle compartment, femoral artery in the lateral compartment and femoral nerve is outside the femoral sheath (From medial to lateral it is **CVAN**).

#### Contents of femoral canal

- Fat
- Lymphatic vessels and
- Lymph node of Cloquet or Rosenmuller.

**Boundaries of femoral canal**

- **Anterior:** Inguinal ligament
- **Posterior:** Astley Cooper's (iliopectineal) ligament, pubic bone and fascia over the pectineal muscle
- **Medial:** Gimbernat's (lacunar) ligament
- **Lateral:** Thin septum which separates the femoral canal from femoral vein.

**Femoral hernia is a hernia through the femoral canal.***History*

- Among hernias in females, indirect inguinal hernia is the most common. But, incidence wise, femoral hernia is more common in middle aged and elderly females than in males. (**Always remember:** Hernias more common in females are femoral, obturator and incisional hernia)
- 20% are bilateral and multiparity is a risk factor
- It is more common on right side  
(**Always remember: Mnemonic – LIMBS** for hernias more common on left side – lumbar, internal paraduodenal, male obturator hernia (female obturator is again more common on right side), bochdalek hernia and sliding hernia)
- Strangulation can be the first presentation in 40% cases. Also, Richter variety is more common in femoral hernias.

**Variants of hernia in the femoral region:**

- **Classic femoral hernia** – through the femoral canal
- **Hasselbach's hernia** – lateral to femoral artery
- **Narath's hernia** – arises from deep to femoral vessels and is associated with congenital hip dislocation
- **Cloquet's hernia** – arises from deep to pectineal fascia
- **Laugier's hernia** – occurs through a gap in the lacunar ligament
- **Beclard's hernia** – occurs through the saphenous opening.

**Coverings**

- Peritoneum
- Extraperitoneal tissue
- Fascia transversalis
- Cribriform fascia covering the saphenous opening
- Superficial fascia
- Skin.

**Examination findings**

- The sac lies below and lateral to the pubic tubercle and is retort shaped
- It is partially reducible and cough impulse is usually absent
- Obstruction and strangulation are more common
- On Ziemann test, the impulse is felt on the ring finger
- On invagination test after reduction of the swelling, no cough impulse is felt.

**Treatment**

- Surgery is the treatment of choice
- Principles include herniotomy followed by closure of the femoral ring
- **Approach:**
  - **McEvedy:** Supra inguinal incision
  - **Lotheissen:** Inguinal incision
  - **Lockwood:** Sub-inguinal incision
  - **Henry procedure:** Low midline extraperitoneal approach.

## GASTROINTESTINAL ONCOSURGERY

- *In this section, I am giving the important SNs on GI malignancies that have been asked or can be asked and points for rapid revision have been added for each malignancy.*
- *This section will help in rapid accumulation of necessary knowledge for each of these cancers.*
- *All management outlines are according to AJCC 7th edition.*

**Q79. Enumerate the risk factors for esophageal cancer.****Ans.**

Adenocarcinoma	Squamous cell carcinoma
<ul style="list-style-type: none"> <li>• Barrett's esophagus (40 times risk)</li> <li>• Obesity</li> <li>• Scleroderma</li> <li>• Diet deficient in fruits and vegetables and high in animal protein and cholesterol.</li> </ul>	<ul style="list-style-type: none"> <li>• Tylosis palmaris et plantaris</li> <li>• Plummer Vinson syndrome</li> <li>• Esophageal diverticula</li> <li>• Bulimia, Vit. A, zinc, molybdenum deficiency</li> <li>• Alcohol, smoking, human papilloma virus</li> <li>• High nitrates, nitrites, nitrosamines, smoked opiates and fungal toxins in pickles</li> <li>• Mucosal damage due to lye, hot liquids, chronic achalasia and radiation.</li> </ul>

**Q. Discuss the management of a patient of esophageal carcinoma.****Ans.**

- **Most common type** is squamous cell carcinoma esophagus.
- **Most important symptom** is progressive dysphagia – first to solids and progressively to semisolids and liquids and finally even saliva.

**Investigations**

- **Upper GI endoscopy with/out EUS and Biopsy** – role of EUS is only in T1/T2 lesions when it can change the management plan. It has no use in proven T3/T4 lesions
- **Double contrast barium swallow** for looking at distal margin of lesion. It has more role in palliative management for planning endoscopic interventions when necessary. It is also useful when endoscopy cannot be performed – scope cannot traverse < 13.5 mm diameter area

- **CECT chest and abdomen**
- **Best staging investigation** – Endoscopy with/out EUS + PET-CT > EUS + CT.

**Management outline:**

**AJCC 7th edition staging**

*Primary tumor (T)*

- **TX:** Primary tumor cannot be assessed
- **T0:** No tumor invasion
- **Tis:** High grade dysplasia
- **T1:** Tumor invades lamina propria, muscularis mucosae or submucosa
  - **T1a:** Tumor invades lamina propria or muscularis mucosa
  - **T1b:** Tumor invades submucosal
- **T2:** Tumor invades muscularis propria
- **T3:** Tumor penetrates adventitia
- **T4a:** Resectable tumor that invades pleura, pericardium or diaphragm
- **T4b:** Unresectable tumor that invades adjacent structures such as vertebra, aorta, trachea, etc.

*Regional lymph nodes (N)*

- **NX:** Regional lymph nodes cannot be assessed
- **N0:** No involvement
- **N1:** Metastasis in 1 to 2 regional lymph nodes
- **N2:** Metastasis in 3 to 6 regional lymph nodes
- **N3:** Metastasis in more than 7 lymph nodes

*Distant metastasis (M)*

- **M0:** No distant metastasis
- **M1:** Distant metastasis

**Treatment for TNM stages**

<b>T1 a N-</b>	Endoscopic mucosal resection /endoscopic mucosal dissection Esophagectomy if margin comes positive after repeated attempts
<b>T1b N-</b>	Esophagectomy. EMR/EMD has high chance of failure
<b>T2N-</b>	Esophagectomy
<b>T2N+, T3, T4</b>	Neoadjuvant concurrent chemoradiation [Paclitaxel and carboplatin in SCC and Epirubicin, cisplatin, 5-Fluorouracil (ECF) in adenocarcinoma for 4 to 5 cycles with 45 gray radiation in 25 fractions] followed by evaluation after 6 weeks and then surgery. This is followed by adjuvant chemoradiation
<b>Metastatic</b>	Palliative stenting and if stenting not possible then surgical bypass in good functional reserve patients and feeding gastrostomy or jejunostomy in poor functional reserve patients

**Q. Enumerate the surgical options for esophagectomy and its reconstruction.**

**Ans. Factors affect surgical decision making:**

- Surgical approach

- Type and position of the replacement conduit
- Location of the anastomosis and anastomotic technique

**Surgical approach**

First esophagectomy done by Torrek

**Transhiatal esophagectomy (first done by Denk, popularised by Orringer)**

- It is the safest esophageal resection technique
- Two incisions, left neck and abdomen
- **Advantages:** Decreased anastomotic leak, less morbid if leak does occur. Reduced operative times, less blood loss and fewer cardiorespiratory complications
- **Disadvantages:** Higher rate of postoperative strictures, injury to great vessels, airway structures, and inability to perform a complete lymph node dissection.

**Transthoracic esophagectomy (Ivor lewis approach)**

- Two incisions, right chest and abdomen
- Complications includes pneumonia, effusions, respiratory failure, atrial fibrillation, and myocardial ischemia
- **Advantages:** The anastomotic leak is least likely to occur
- **Disadvantages:** When an anastomotic leak does occur, it may be difficult to control and can lead to an intrathoracic infection, sepsis, and death.

**En bloc esophagectomy (McKeown approach)**

- Three incisions—left neck, right chest, and abdomen
- Most morbid of all the procedures.

**Vagal-Sparing esophagectomy**

- The technique varies only in the method of removing the esophagus without severing the vagus nerves.

**Minimally Invasive esophagectomy**

Thoracoscopy and laparoscopy are being increasingly used for the treatment of carcinoma esophagus.

There is no significant difference in mortality/morbidity and survival outcomes in the patients managed with transhiatal or transthoracic approach. An intrathoracic esophagogastric anastomosis has a better chance of healing.

**Replacement conduits**

Stomach (based on right gastric and right gastroepiploic), colon, jejunum are all used as conduits after esophagectomy. Placed through posterior mediastinum, anterior mediastinum or substernal routes.

**Lymphadenectomy**

2 field lymphadenectomy (intra-abdominal and intrathoracic) is adequate and 3 field (2 field + cervical) has no added benefit.

**Q80. Discuss the pathology of carcinoma stomach.**

**Enumerate the risk factors for carcinoma stomach.**

**Write a note on etiopathogenesis of carcinoma stomach.**

**Discuss staging of gastric cancer.**



**Ans.**

### **Pathogenesis**

- **Risk factors** (Mn: SOMENG)
  - Social – low social class
  - Occupational – rubber, coal workers
  - Medical – prior gastric surgery, *H. pylori*, Epstein Barr virus, gastric atrophy, gastritis, adenomatous polyps, male gender, Menetrier's disease
  - Environmental- smoking, smokes and salted food, lack of refrigeration, poor drinking water (well water)
  - Nutritional – low fat or protein consumption, high nitrate or complex carbohydrate consumption
  - Genetic- Li-Fraumeni syndrome, HNPCC, family history, blood group A, Pernicious anemia
- Correa model for adenoma carcinoma sequence in pathogenesis of stomach cancer.

**Site – pylorus of the stomach is the most common site** > body > cardia

Entire stomach involvement in 7% called linitis plastica

### **Macroscopic appearance**

- Proliferative cauliflower growth
- Ulcerative (m.c.) with elevated edges, surrounding edema and flattened mucosal folds
- Colloid/mucoid type has a gelatinous appearance wherein cancer cells are surrounding a core of accumulated colloid and mucin.
- Linitis plastic – extensive infiltration of tumor cells into all the layers without a growth in the lumen with proliferation of fibrous tissue in the submucosa called mother of pearl appearance. Mucosa may appear normal. It has two varieties – generalized and localized (m.c. in pylorus).

### **Histologic types**

- Adenocarcinoma (m.c.) is of two types intestinal and diffuse. Mucinous and colloid differentiation can occur.
- Squamous cell carcinoma is m.c. in cardia
- Adenoacanthoma is mixed adenocarcinoma and squamous carcinoma
- Lymphoma, leiomyoma, GIST, carcinoid are other histological subtypes in stomach cancer all of which are submucosal masses.

**Most common histological markers for development of stomach cancer** – intestinal metaplasia type 3 and dysplasia

**Tumor markers** of carcinoma stomach – Ca 19-9, Ca 72-4, CEA.

### **Pathological classifications of carcinoma stomach**

- **Lauren classification** – intestinal and diffuse
- **Borrmann classification**
  - Polypoidal or fungating
  - Ulcerated lesion with elevated borders
  - Ulcerated lesion infiltrating gastric wall
  - Linitis plastic
  - Unclassifiable

- **Japanese classification of early gastric cancer**
  - Exophytic
  - A. elevated
    - B. flat
    - C. depressed but not ulcerated
  - Ulcerated upto muscularis propria
- **Pathological staging of carcinoma stomach specimen** according to AJCC 7th edition.

*Primary tumor (T)*

- **TX:** Primary tumor cannot be assessed
- **T0:** No tumor invasion
- **Tis:** Carcinoma in situ: Intraepithelial tumor without invasion into lamina propria
- **T1:** Tumor invades lamina propria, muscularis mucosae or submucosa
  - **T1a** Tumor invades lamina propria or muscularis mucosa
  - **T1b** Tumor invades submucosal
- **T2:** Tumor invades muscularis propria
- **T3:** Tumor penetrates subserosal connective tissue without invasion of visceral peritoneum or adjacent structures. T3 tumor also includes those extending into the gastrocolic or gastrohepatic ligaments, or into the greater or lesser omentum, without perforation of visceral peritoneum covering these structures into adjacent structures
- **T4:** Tumor invades serosa (visceral peritoneum) or adjacent structures

*Regional lymph nodes (N)*

- **NX:** Regional lymph nodes cannot be assessed
- **N0:** No involvement
- **N1:** Metastasis in 1 to 6 regional lymph nodes
- **N2:** Metastasis in 7 to 15 regional lymph nodes
- **N3:** Metastasis in more than 15 lymph nodes

*Distant metastasis (M)*

- **M0:** No distant metastasis
- **M1:** Distant metastasis.

**Grading**

- Gx
- G1 – well differentiated
- G2 – moderately differentiated
- G3 – poorly differentiated
- G4 – undifferentiated

Pathological prognostic factors – depth of tumor invasion and lymph node status.

- Q. Write a note on investigations and management of carcinoma stomach. Discuss the TNM staging of carcinoma stomach and outline its management.**

**Ans.**

- **Most common symptom** – abdominal pain.

- Other important presentations include anorexia, vomiting, GOO, weight loss. It can also present with paraneoplastic syndromes such as Trousseau's syndrome (Migratory thrombophlebitis) or neuropathies.

### Investigations

- **Investigation of choice for diagnoses** – UGI endoscopy and biopsy
- **For staging** – CECT abdomen
- **Diagnostic laparoscopy** is done when CT shows perigastric ascites which can be due to peritoneal disease/hypoproteinemia/Inflammatory cause.

### Management outline

T1, T2 node negative	Gastric resection procedure*
<b>T2N+, Any T3,T4 and any medically fit unresectable Nonmetastatic tumor</b>	Neoadjuvant chemotherapy [ECF as above or EOX (Oxaliplatin and epirubicin) +/- Capecitabine- 2 to 3 cycles] followed by evaluation of response followed by surgery if resectable followed by adjuvant chemoradiotherapy or consolidation chemotherapy
<b>Metastatic disease</b>	Palliation for gastric outlet obstruction, pain, UGI bleed if present by bypass procedure if patient is good performance status or Feeding tubes distal to the tumor for poor performance status.

\* Extended gastrectomy including distal esophagus and D2 lymphadenectomy for tumors in proximal third of stomach, total gastrectomy with D2 lymphadenectomy for all others. Intestinal type distal third tumors can be managed with subtotal gastrectomy with D2 dissection rather than total gastrectomy.

**Indications of splenectomy with gastrectomy** – Direct involvement of spleen or splenic vessels, size > 5 cm, perineural and perilymphatic invasion.

**D1** – Stations 3 to 6 are to be resected.

**D2** – Stations 1 to 8, 11 are to be resected.

**D3** – Stations 1 to 12 are to be resected.

D2 is better than D1. There is no oncological benefit between D2 and D3 and D2 is enough. Reconstruction can be with **Cuschieri pouch** (interposed jejuna pouch) if GE junction is preserved or with **Merendino procedure** (Isoperistaltic jejuna interposition) if GE junction is also resected.

### Q81. Enumerate the classification and management of GE junction tumors.

**Ans. Types**

*Siewert classification*

- Type 1—Located in esophagus and within 1 to 5 cm of GEJ. Managed like Esophagus cancer
- Type 2—Within 1 cm above GEJ to 2 cm below GEJ. Managed like stomach cancer
- Type 3—Located in stomach and within 2 to 5 cm of GEJ. Managed like stomach cancer.

### Investigations

- UGI endoscopy and biopsy
- CECT chest and abdomen.

**Management outline**

Type 1 managed like esophageal cancer as above	Transthoracic approach preferred with abdominal and lower mediastinal lymph node clearance. Trend is towards en-bloc esophagectomy with 2 field nodal clearance
Type 2 and 3 are managed like gastric cancer as above	Extended gastrectomy with D2 lymphadenectomy with transhiatal esophagectomy for 5 cm margin with mediastinal and paraaortic lymph node clearance

Reconstruction is also done as above for esophagus and/or stomach as per resection extent.

**Q82. Enumerate the risk factors for small bowel adenocarcinoma.**

**Ans.** Most common malignancy of small bowel is carcinoid > adenocarcinoma

**Risk factors are as follows:**

- Crohn's disease
- Celiac disease (Gluten sensitive enteropathy)
- FAP, HNPCC
- Peutz Jeghers syndrome
- Biliary diversion (Prior cholecystectomy)
- Smoking, heavy alcohol consumption and consumption of red meat or salt cured foods are controversial risk factors.

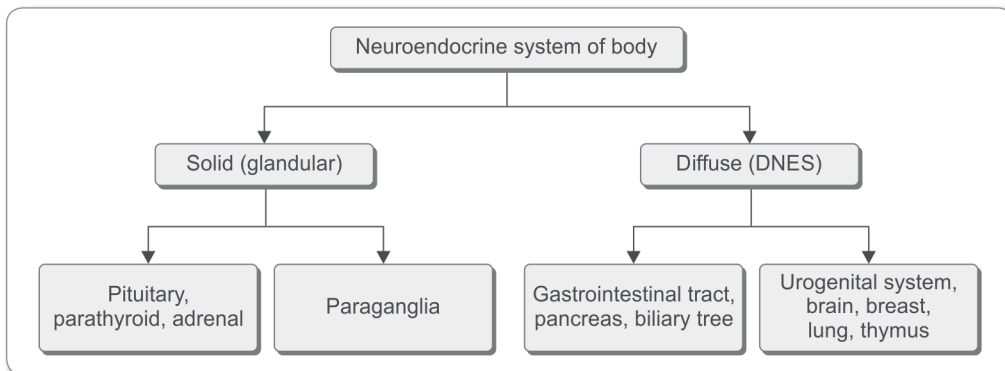
**Q83. What is carcinoid tumor ? Discuss its clinical features and management.**

**What is carcinoid syndrome ? Discuss its management.**

**Discuss gastric carcinoid tumors in brief.**

**Ans.**

- Carcinoid tumors are neuroendocrine tumors arising from kulchitsky cells
- Also called GEP-NETS- gastro entero pancreatic neuroendocrine tumors
- Also called well differentiated neuroendocrine neoplasm of luminal GI tract or serotoninomas.



**WHO classification of neuroendocrine tumors**

Three main categories:

- Well-differentiated neuroendocrine tumours, further subdivided into tumors with benign and those with uncertain behavior
- Well-differentiated (low grade) neuroendocrine carcinomas with low-grade malignant behavior
- Poorly differentiated (high grade) neuroendocrine carcinomas, which are the large cell neuroendocrine and small cell carcinomas.

**Incidence** in different organs : bronchus > intestine > rectum > appendix > colon > stomach.

**GI carcinoids**

Largest single endocrine organ is GI tract.

Can arise from foregut, midgut and hind gut.

- Foregut and hindgut tumors are argyrophilic (stain with silver stain on adding reducing agent)
- Midgut tumors are argentaffinic (directly stain with silver stain).

**Markers**

- Chromogranin A – Essential for generation of vesicles and dense core granules
- Synaptophysin and neuron specific enolase – cellular antigens.

**Endodermal origin**

2nd most common malignancy of GI tract (1st is colorectal cancer).

**Risk factors**

- Parental history
- Sibling with carcinoid
- Long standing diabetes (associated with gastric carcinoid in women)
- Parent with carcinoma brain, breast, liver and endocrine/urinary system
- Organic solvents and lead paints are associated with small bowel carcinoids.
- Syndromic associations : MEN 1 and VHL with PNET, neurofibromatosis 1, Tuberous sclerosis.

**Grading**

Grade	Differentiation	Mitoses/hpf	Ki- 67 index
Low	Well differentiated	<2 AND	< or = 2
Intermediate	Well differentiated	2- 20 OR	3 – 20
High	Poorly differentiated	>20 OR	> 20

**Carcinoid syndrome**

Ten per cent (10%) or less of carcinoids, primarily some midgut carcinoids, secrete excessive levels of a range of hormones, most notably serotonin (5-HT) or substance P, causing a constellation of symptoms called carcinoid syndrome:

- Flushing – episodic, affects face and torso
- Diarrhea – secretory
- Asthma or wheezing

- Congestive heart failure (CHF)
- Abdominal cramping
- Peripheral edema
- Heart palpitations

A carcinoid crisis with profound flushing, bronchospasm, tachycardia, and widely and rapidly fluctuating blood pressure can occur if large amounts of hormones are acutely secreted, which is occasionally triggered by factors such as diet, alcohol, surgery chemotherapy, embolization therapy or radiofrequency ablation.

Chronic exposure to high levels of serotonin causes thickening of the heart valves, particularly the tricuspid and the pulmonic valves, and over a long period can lead to congestive heart failure. However, valve replacement is rarely needed.

The excessive outflow of serotonin can cause a depletion of tryptophan leading to niacin deficiency, and thus pellagra, which is associated with dermatitis, dementia, and diarrhea.

#### *Clinical presentation*

- **m.c.-** cramping abdominal pain
- Can also present as intermittent small intestinal obstruction or GI bleeding.

#### *Lab evaluation*

- Urinary 5- HIAA – most useful in midgut carcinoids
- Serum chromogranin A
- Blood serotonin levels.

#### *Imaging*

- CECT is imaging modality of choice as it also shows liver involvement, retroperitoneal and mesenteric involvement, and lymph nodes.
- MRI is most effective for liver metastasis.
- Endoscopic ultrasound and guided biopsy
- Octreoscan (somatostatin receptor scintigraphy)

#### *Site specific points*

#### **Gastric carcinoids [arises from ECL cells (secrete histamine)]**

- **Type 1**
  - M.C. – 75%
  - Associated with chronic atrophic gastritis
  - Small, multiple, well differentiated
  - Prolonged survival
  - **Management:**
    - < 1 cm—Observation and surveillance
    - 1 – 2cm—Endoscopic mucosal resection
    - < 2 cm—Antrectomy with local resection with lymphadenectomy for positive nodes
- **Type 2**
  - Associated with ZES and MEN 1
  - 5%, often have distant disease

- Small, multiple, well differentiated
- Medical management or pancreaticoduodenectomy or pancreas preserving resection are options.
- **Type 3**
  - Large, singular, not associated with hypergastrinemia
  - More morbid, worse prognosis
  - Produces atypical carcinoid syndrome (5- HT)
  - Manage like gastric adenocarcinoma
- **Type 4**
  - Non ECL type
  - Paneth cell hyperplasia
  - More invasive, metastatic potential
  - Located anywhere in stomach
  - Manage like gastric adenocarcinoma

**Small bowel** (It is the most common small bowel tumor)

- Serotonin producing tumors
- m.c. site is ileum
- CT – mesenteric tumor with radiating spokes

Preoperative upper and lower GI endoscopy should be done as 30% have metastatic lesion and 30% have associated other malignancies

Segmental resection with wide mesenteric lymphadenectomy is the treatment.

### Colorectal

Colon	Rectum
Large >5 cm	Small <1 cm
Metastatic in 40%	Localized 80%
Cecum is m.c. site	
EUS and biopsy is test of choice	
Manage like adenoca.	Manage as per rectal resection procedures if tumor invades lamina propria, mesorectal lymph nodes, ulceration, lymphovascular invasion, or size > 2 cm

### Appendix

For appendiceal tumors, indications for right hemicolectomy include:

- 2 cm
- Mesoappendix invasion
- Mixed histology
- Base invasion
- Lymphovascular invasion
- Intermediate to high grade

**Metastatic disease** – pancreatic (64%) > cecal (44%) > jejunoileal (30%)

*Medical management*

- Octreotide
- Sunitinib
- Everolimus
- Interferon alfa
- Alkylating agents

Surgery for resectable liver metastasis or when > 90% tumor can be resected.

*Transplantation*

- Isolated hepatic metastatic disease
- Complete resection of primary possible
- Low proliferative index
- Absence of hepatomegaly
- Primary should not be pancreatic or rectal.

*Perioperative care*

- Avoid carcinoid crises by giving octreotide 200 microgram s/c tds for few weeks before surgery and 50 microgram/hour infusion intraoperative.
- Rule out carcinoid heart disease (tricuspid > pulmonary valve).
- Consider prophylactic cholecystectomy (factors octreotide and hepatic artery embolisation in management can cause gallstones and biliary tree complications).

**Q84. Write a note on gastrointestinal stromal tumor.**

**Ans.**

**Most common mesenchymal tumor of GIT**

**Cells of origin** – Interstitial cell of cajal are called interstitial because they have properties between nerve cells and muscle cells in having very few contractile elements and greater proportion of mitochondria. They normally play a role in propagation of intrinsic slow wave gut peristalsis.

- **Extra GIST** – Omentum, genitourinary system, portal vein, pancreas
- **Most common site** – stomach > small intestine

**Immunohistochemical markers (both kit and PDGFRA map to ch.4q12)**

- CD 34
- CD 117 (ckit) – tyrosine kinase receptor (exon 11 mutation m.c., others exon 9, 13)
- PDGFRA
- DOG1 (discovered on GIST)
- Exon 17 mutation is more commonly associated with familial GIST
- All GISTs have some ability to metastasize and should never be considered truly benign

Most important factors in governing recurrence free survival include size, number of mitoses and location.



**Risk stratification**

Risk	Size	Mitoses
Very low risk	<2 cm	< 5/50 hpf
Low risk	2–5 cm	< 5/50 hpf
Intermediate risk	5–10 cm	< 5/50 hpf
	< 5 cm	6–10 / 50 hpf
High risk	> 5 cm	>5 / 50 hpf
	>10 cm	Any mitotic rate
	Any size	>10/50 hpf

**Presentation**

- Asymptomatic (incidental finding on tests)
- Palpable mass, GI bleeding, anemia, abdominal pain.

**Imaging**

- CECT – submucosal mass with smooth borders and rounded appearance or exophytic lobulated lesion
- Endoscopy, EUS with guided biopsy can be used.

**Management**

- Surgery is the treatment of choice
  - Macroscopic clear margin is the aim as the presence of microscopic positive margin does not affect prognosis
  - In gastric GISTs partial gastrectomy gives the same progression free survival as total but with less morbidity
  - Minimally invasive approach is commonly being used now as given by **Privette et al study for gastric GISTs (2007)**.

Type 1	Greater curvature and fundus	Lap. Stapled partial gastrectomy
Type 2	Prepyloric region and antrum	Lap. Distal gastrectomy
Type 3	Lesser curvature and GEJ	Lap. Transgastric resection

**Role of biologic therapy***Imatinib mesylate*

- Adjuvant in all patients with tumor > 3 cm for 3 years
- Neoadjuvant in all patients with tumor > 10 cm
- All metastatic, recurrent and locally invasive GISTs
- Side effects : periorbital and peripheral edema, diarrhea, fatigue, mild hypertension.

**Sunitinib** given in imatinib refractory cases or in patients not tolerating imatinib.

*Recent studies show*

- Exon 11 mutations respond well to imatinib.
- Exon 9 mutants are less sensitive to imatinib and therefore require higher doses.

- M.c. PDGFRA mutation is exon 18 and it is resistant to imatinib.
- PDGFRA mutation is more commonly associated with gastric GISTs, has epithelioid morphology and is less malignant.

### Follow up

- Clinical evaluation and CT at 6 months interval till 2 years.
- Then annually till 5 years.

### Atypical presentations

#### *Pediatric GIST*

- More often epithelioid
- More indolent course
- m.c. symptom is anemia
- m.c. site is stomach
- More favorable prognosis
- Role of IGFIR being considered.

### Connections with syndromes

- Carney's triad
  - GIST, paraganglioma and pulmonary chondroma
  - Has female predilection with age < 30 yrs
- Also associated with neurofibromatosis 1
- Syndromic GISTs (Carney and NF 1) lack kit and PDGFRA mutations.

## Q85. Enumerate the risk factors for colorectal cancer.

Ans.

1. **Premalignant colon conditions** (All hereditary and hamartomatous diseases are autosomal dominant except Turcot syndrome which is autosomal recessive)

Hereditary polypoid diseases	FAP (Ch.5)	Classic FAP
		Attenuated FAP
		MYH associated polyposis
		Gardner syndrome
		Turcot syndrome ( <b>AR</b> )
		Attenuated FAP
Hereditary nonpolypoid diseases	HNPCC	Lynch type 1 (only colon)
		Lynch type 2
		Muir-torre variant
Inflammatory bowel diseases	Ulcerative colitis Crohn's colitis	
Nonhereditary polypoid lesions	Hamartomatous*	Juvenile polyposis syndrome (Ch. 18) ( <b>m.c.</b> )
		Peutz-jeghers syndrome (Ch. 19p)

Contd...

Contd...

	<b>Adenomatous</b>	Tubular adenoma
		Tubulovillous adenoma
		Villous adenoma (most chances of malignancy)

\*Hamartomatous syndromes not associated with gastrointestinal malignancy include Cowden syndrome, Conkhrite Canada syndrome and Banayan - Ruvalcaba - Myhre - Smith syndrome

## 2. Risk factors

- Dietary – alcohol, low fat diet, high animal fat diet
- Ureterosigmoidostomy, radiation, smoking
- Acromegaly, *Streptococcus bovis* bacteremia
- Hereditary syndromes – FAP, HNPCC
- Inflammatory bowel disease – ulcerative colitis and Crohn's disease.

## Q. Discuss the screening criteria and methods in colorectal cancer.

### Ans. Average risk individuals

Screening beginning at age 50 years.

- **Colonoscopy** is the preferred modality
  - False negative rate is 27% for adenomas  $\leq 5$ mm and 6% for lesions  $\geq 10$ mm
  - Done every 10 years.
- **FOBT**
  - Annually, 2 samples from each of 3 consecutive stools
  - If positive, colonoscopy should follow.
- **Flexible sigmoidoscopy**
  - Reduction in colorectal cancer incidence in portion of the colon examined, and decreased mortality between 59 to 80%
  - Prevalence of proximal neoplasia increases with age, therefore colonoscopy may be better suited for screening in patients older than 60 years
  - Recommended every 5 years.
- **FOBT and flexible sigmoidoscopy**
  - No evidence that combination of annual FOBT and flexible sigmoidoscopy every 5 years reduces CRC mortality
  - 70.3% of patients with advanced neoplasia were identified by the use of, sigmoidoscopy alone and 75.8% with the addition of FOBT.
  - Combination of FOBT and flexible sigmoidoscopy every 5 years may be considered. Addition of FOBT have minimal benefit.
- **Double-contrast barium enema (DCBE)**
  - Diagnostic sensitivity inferior to colonoscopy and lacks therapeutic capability
  - Not recommended for screening
  - If used, it should be done every 5 years.
- **Virtual colonoscopy (VC)**
  - Sensitivity of 55 to 100% and specificity of 94 to 98% for detection of polyps  $\geq 10$ mm
  - May detect extra-colonic findings.

**Individuals with family history of CRC or adenomatous polyps**

- **First degree relatives with CRC diagnosed age <60 years**  
Colonoscopy at age 40 years or 10 years younger than affected relative. Repeat every 3 to 5 years.
- **First degree relatives with CRC diagnosed age ≥60 years**  
Colonoscopy at age 40 years and repeat every 10 years.
- **First degree relatives with adenomatous polyp diagnosed age <60 years**  
Colonoscopy at age 40 years or 10 years younger than affected relative. Repeat every 5 years.
- **First degree relatives with adenomatous polyp diagnosed age >60 years**  
Colonoscopy age individualized and follow up as for average risk patients.
- **Second- or third degree relative with cancer or polyps**  
As for average risk patients.

**Other screening guidelines for intermediate risk and high risk population**

- **In patient with a simple adenoma <1cm excised** – Colonoscopy at 3 month than 1 year than as per average risk individual if findings are normal.
- **If adenoma has malignant features** – Do colonoscopy at 1 month, 3 months, than as per guidelines for the average risk individuals
- **If personal history of cancer cure** - Tertiary prevention screening is colonoscopy at 1 year, 3 year and 5 years. Then, 5 yearly
- **FAP** – Yearly colonoscopy since 10 to 12 years age (puberty)
- **HNPCC** – Yearly or once in 2 years colonoscopy since age of 21 years
- **Patients with pancolitis** – Start colonoscopy since 8 years of disease once in 2 years till age of 40, than annually.
- **Patients with left sided colitis** - Start colonoscopy since 8 yrs of disease once in 2 years till age of 40, than annually.

**Q. Write a note on pathology of carcinoma colon.**

**Discuss pathogenesis of carcinoma colon.**

**Write the staging systems of colorectal cancer.**

**Write a note on DUKE'S staging for colorectal cancer.**

**Ans. Pathogenesis** is given by adenoma carcinoma sequence also called **Fearon vogelstein model**

**Macroscopic types**

- Annular
- Tubular
- Ulcerative
- Proliferative/cauliflower.

**Histology**

- Adenocarcinoma with varying degrees of differentiation as follows:
  - In situ

- Mucinous type (when > 50% cells are mucus cells)
- Signet ring cell type (when > 50% cells are of this type)
- Adenosquamous differentiation
- My undergo colloid/mucinous degeneration
- Squamous cell carcinoma
- Lymphoma, Leiomyoma, GIST.

**Grading**

- Gx
- G1—well differentiated
- G2—moderately differentiated
- G3—poorly differentiated
- G4—undifferentiated

**Pathological prognostic markers**

- Circumferential resection margin
- CEA
- Microsatellite instability
- KRAS analysis
- Tumor deposits
- R1/R2 resection
- Lymphovascular invasion.

**Pathological staging of colorectal cancer specimen**

AJCC 7th edition staging

*Primary tumor (T)*

- **TX:** Primary tumor cannot be assessed
- **T0:** No evidence of primary tumor
- **Tis:** Carcinoma in situ
- **T1:** Tumor invades submucosal
- **T2:** Tumor invades muscularis propria
- **T3:** Tumor invades subserosa or beyond (without other organs involved)
- **T4:** Tumor invades adjacent organs or perforates the visceral peritoneum

*Regional lymph nodes*

- **NX:** Regional lymph nodes cannot be assessed
- **N0:** No involvement
- **N1:** Nodal metastasis to 1 to 3 regional nodes, T1 or T2
- **N2:** Nodal metastasis to 4 or more regional nodes.

*Distant metastasis*

- **M0:** No distant metastasis
- **M1:** Distant metastasis.

<b>Stage 0</b>	Tis N0 M0
<b>Stage I</b>	T1,2 N0 M0
<b>Stage II</b>	T3,4 N0 M0
<b>Stage III</b>	Node positive
<b>Stage IV</b>	Metastatic

In AJCC 7th edition, N1 is subdivided into

- N1A – One node involved
- N1B- 2 or 3 nodes involved
- N1C – Tumor deposits in the subserosa, mesentery, or non peritonealized pericolic or perirectal tissues without regional nodal metastasis

Other classifications include the following:

- **Original Duke's classification**
  - Confined to bowel wall
  - Penetrates bowel wall completely
  - Indicates lymph node metastasis
- Later came to be known as **modified Duke's classification** or Astler Coller classification due to following three modifications (**Mn: KAT**)
  - **Kirklin** divided B into B1 (partially penetrated muscularis propria) and B2 (completely penetrated muscularis propria)
  - **Astler collar** divided C into C1 (lymph node invasion without penetration of entire bowel wall) and C2 (lymph node invasion with penetration of entire bowel wall)
  - **Turnbull** added D to the original Duke classification – distant metastasis.

**Q. Discuss the management of a patient with colon cancer.**

**Ans.**

- **Identification of the site of lesion. Right sided lesion** present more commonly with anemia, weight loss, melena, fatigue, abdominal pain and right iliac fossa lump. It has a cauliflower like growth and is nonobstructing. It has a better prognosis.
- **Left sided colonic malignancy** on the other hand presents with symptoms of progressive obstruction such as decrease in the calibre of stools, alteration in bowel habits or palpable lump. It present as annular, stenosing growth.
- **Investigations include CECT abdomen and pelvis and colonoscopy. Colonoscopy is the gold standard** for diagnoses as well as exclusion of synchronous and metachronous lesions. (Synchronous lesions make appearance within 6 months of the primary lesion and metachronous lesions after that duration).
- **Management outline is as follows:**

<b>TisN0</b>	Endoscopic resection (colonoscopy)
<b>T1N0</b>	Segmental resection
<b>T1-3N0</b>	Resection according to site
<b>Node positive (Stage III)</b>	Resection followed by adjuvant chemotherapy

Contd...

Contd...

<b>Metastasis (Stage IV)</b>	Resection for resectable metastasis (m.c. liver > lungs) + Adjuvant chemotherapy Palliative care for all other patients
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**Indications of adjuvant chemotherapy are as follows:**

- Patient presenting with obstruction/perforation
- Insufficient node sampling (< 12 nodes)
- Perivascular invasion
- Poorly differentiated histology

**FOLFOX –IV** (5-Fluorouracil, leucovorin and Oxaliplatin) is the regimen most commonly used for 12 cycles. Other include FOLFIRI (Irinotecan). Targeted therapy (Cetuximab, bevacizumab, Regorafenib) are used in stage IV disease.

**Q. Write a note on the management outline of rectal cancer.****Ans.**

- **Bleeding per rectum is the most common symptom**
- Sense of incomplete defecation, early morning spurious diarrhea or mucus per rectum are all other symptoms with rectal cancer. Growth at the rectosigmoid junction can present with increasing constipation.
- **Investigation of choice for diagnoses is rigid sigmoidoscopy and biopsy and for staging is pelvic MRI.** TRUS and endorectal coil MRI were considered best for T staging but pelvic MRI has surpassed all other investigations.
- **Management outline is as follows:**

<b>TisN0</b>	Transanal endoscopic microsurgery (TEMS) or local excision
<b>T1,2N0</b>	Local resection Radical resection in patients with poor histologic features or high risk group
<b>T3,4 N0</b>	Neoadjuvant chemoradiation followed by standard resection procedures*
<b>Any Node positive tumor</b>	Neoadjuvant chemoradiation followed by standard resection procedures*
<b>M1 patients</b>	Palliation which may include resection for bleeding, tenemus or pain.

**\*Resection procedures are as follows:**

- **TEMS/Local excision** – T1,2 N0 lesion within 10 cm off anal verge and involving less than 1.3 of the bowel circumference and <4 cm diameter.
- **Anterior resection** – Anastomosis is accomplished with the peritonealised rectum.
- **Low anterior resection** – Anastomosis is accomplished with extraperitoneal rectum after full mobilisation of the rectum and division of the lateral ligaments.
- **Ultralow (Extended low) anterior resection** – Anastomosis constructed at or just above levator ani.

**Indications of neoadjuvant chemoradiation**

- T3,T4 tumors
- Extramural vascular invasion on MRI with any stage of tumor
- Circumferential resection margin (margin of resection) involved by tumor or threatened
- Nodal positive tumors

- **Type 3 or 4 (Low) rectal cancer** (Described below).

<b>Type 1 (supraanal)</b>	>1 cm from anal ring	Low anterior resection
<b>Type 2 (Juxtaanal)</b>	<1 cm from anal ring but not involving sphincter	Partial intersphincteric abdominoperineal excision
<b>Type 3 (Intraanal)</b>	Internal anal sphincter invasion	Total intersphincteric abdominoperineal excision
<b>Type 4 (Transanal)</b>	External and internal sphincter invasion	Abdominoperineal excision of rectum

**Q86. What are the risk factors for carcinoma anal canal. Outline its management.**

**Ans. Risk factors**

- HPV (16, 18, 31, 33)
- HIV or immunosuppression
- Smoking
- Anal receptive intercourse, sexual promiscuity
- Anal intraepithelial neoplasia
- History of vulva or cervical cancer.

**Management**

- Rectal bleeding, pain, Incontinence, change in bowel habits are possible symptoms
- M.C. site of metastasis is lung
- Proctoscopy with biopsy is the investigation of choice
- CECT abdomen and pelvis for staging.

**Treatment for any stage is chemoradiation called NIGRO regimen which is as follows:**

- 5-Fluorouracil 1000 mg/m<sup>2</sup>/24 hrs infusion for 4 days starting on day 1 and repeated on day 28
- Mitomycin C 15 mg/m<sup>2</sup> bolus on day 1 and day 29
- 200 rads/day from day 1 for 15 days to primary tumor, pelvis and inguinal nodes.

**APER is done for residual tumor or nonresponse to chemotherapy.**

**Q87. What are the risk factors for hepatocellular carcinoma.**

**Ans.**

<b>Cirrroses</b>	HBV, HCV, alcohol, autoimmune, cryptogenic, NAFLD, Primary biliary cirrroses
<b>Metabolic</b>	Hemochromatosis, tyrosinemia, Alpha 1 antitrypsin deficiency, ataxia telangiectasia, galactosemia, citrullinemia, allagile syndrome, Wilson disease, orotic aciduria, porphyria cutanea tarda
<b>Environmental</b>	Thorotrast, smoking, aflatoxin, androgenic steroids

**Q. Outline Management of a case of hepatocellular carcinoma.**

**Ans. Surveillance (Abdominal ultrasound every 6 months)**

Following high risk patients for developing HCC are called for surveillance:

- Cirrhotic patients, child-Pugh stage A and B
- Cirrhotic patients, child-Pugh stage C awaiting liver transplantation



- Non-cirrhotic HBV carriers with active hepatitis or family history of HCC
- Non-cirrhotic patients with chronic hepatitis C and advanced liver fibrosis F3

#### **In cirrhotic patients,**

- Nodules less than 1 cm—Followed every 4 months the first year and with regular checking every 6 months thereafter
- Nodules of 1 to 2 cm—Diagnosed based on results of 2 imaging techniques (see criteria below) or 1 imaging technique with biopsy-proven pathological confirmation
- Nodules more than 2 cm - Diagnosed based on typical features on one imaging technique. And biopsy is done only in cases of uncertainty.

#### **Diagnosis**

- Diagnosis of HCC is based on non-invasive criteria or pathology
- Non-invasive criteria can only be applied to cirrhotic patients and are based on imaging techniques obtained by 4-phase multidetector CT scan or dynamic contrast-enhanced MRI
- Diagnosis should be based on the of the typical hallmark of HCC (**hypervascular in the arterial phase with washout in the portal venous or delayed phases**)
- While one imaging technique is required for nodules beyond 1 cm in diameter, a more conservative approach with 2 techniques is recommended in suboptimal settings

#### **Staging systems**

The BCLC staging system is recommended for prognostic prediction and treatment allocation.

#### **Treatment**

- Treatment allocation is based on the BCLC allocation system.

#### **1. Resection**

*Patient selection –*

- Stage 0 BCLC (very early) –
- In situ lesion/single tumor < 2 cm with no nodes/metastases
- With excellent performance status (ECOG 0) and Child Pugh A
- With normal portal pressure, bilirubin and no associated diseases.

*Anatomical resections with 2 cm margin*

Neo-adjuvant or adjuvant therapies do not improve outcome of patients treated with resection (or local ablation).

#### **2. Liver transplantation**

*Patient selection -*

- Early stage A BCLC –
- Single tumors less than 5 cm or  $\leq 3$  nodules  $\leq 3$  cm (Milan criteria) not suitable for resection
- ECOG 0, child pugh A- B
- Raised portal pressure/ bilirubin but, still no associated diseases.
  - Neo-adjuvant treatment can be considered for loco-regional therapies if the waiting list exceeds 6 months.
  - Living donor liver transplantation is an alternative option in patients with a waiting list exceeding 6 to 7 months.

### 3. Local ablation

#### *Patient selection -*

Same patients that were in resectable or transplant list but, WITH associated diseases

- **Options** : Chemical ablation (Ethanol, acetic acid) and thermal ablation (Radiofrequency ablation, microwave ablation, laser ablation and cryoablation)
- Radiofrequency ablation is recommended in most instances as the main ablative therapy in tumors less than 5 cm due to a significantly better control of the disease
- Ethanol injection is recommended in cases where radiofrequency ablation is not technically feasible (around 10-15%)
- In tumors <2 cm, BCLC 0, both techniques achieve complete responses in more than 90% of cases with good long-term outcome.

### 4. Chemoembolization and transcatheter therapies

#### *Patient selection -*

- BCLC stage B, multinodular asymptomatic tumors without vascular invasion or extra-hepatic spread
- ECOG 0, child pugh A-B
  - **Options** : Transarterial chemoembolization (TACE), radioembolization, transarterial embolization (TAE)
  - Chemoembolization is discouraged in patients with decompensated liver disease, advanced liver dysfunction, macroscopic invasion or extrahepatic spread.

### 5. Systemic therapy

#### *Patient selection -*

- Stage C BCLC – Multinodular tumors with portal invasion, node positive or metastatic
- With ECOG 1, 2 and child pugh A to B
  - Sorafenib is the drug of choice
  - There is no available second-line treatment for patients with intolerance or failure to sorafenib
  - In circumstances, radiotherapy can be used to alleviate pain in patients with bone metastasis.

### 6. Palliative care

#### *Patient selection -*

- Stage D BCLC
  - Management of pain, nutrition and psychological support
  - They should not be considered for participating in clinical trials

**Q88. What are the risk factors for carcinoma gallbladder? Enumerate them.**

**Ans.**

**Risk factors for carcinoma gallbladder are as follows:**

- Gallstones especially > 3 cm
- Anomalous pancreaticobiliary junction
- Adenomatous polyp
- Porcelain gallbladder

- Choledochal cyst
- Primary sclerosing cholangitis
- *Salmonella typhi* infection
- Obesity
- *Clonorchis sinensis* infection.

**Q. Discuss the management of a patient with carcinoma gallbladder.**

**Write the staging and stage wise management outline for carcinoma gallbladder.**

**Ans.**

**Diagnosis**

- First investigation—Ultrasonography
- Best investigation for staging—CECT
- Indications of biopsy
  - Unresectable carcinoma gallbladder
  - Before enrolment into clinical trials for neoadjuvant chemotherapy
  - Suspicion in diagnoses.

**Staging**

AJCC staging 7th edition

*Primary tumor (T)*

- **TX:** Primary tumor cannot be assessed
- **T0:** No evidence of primary tumor
- **Tis:** Carcinoma in situ
- **T1:** Tumor invades lamina propria or muscularis layer
  - **T1a** Tumor invades lamina propria
  - **T1b** Tumor invades muscularis layer
- **T2:** Tumor invades perimuscular connective tissue; no extension beyond serosa layer or into the liver
- **T3:** Tumor perforates the serosa or directly invades the liver or one other adjacent structures or organs such as stomach, duodenum, colon, pancreas, omentum or extrahepatic bile ducts
- **T4:** Tumor invades main portal vein, hepatic artery or invades two or more extrahepatic structures or organs.

*Regional lymph nodes (N)*

- **NX:** Regional lymph nodes cannot be assessed
- **N0:** No involvement
- **N1:** Nodal metastasis to nodes along cystic duct, common bile duct, hepatic artery or portal vein
- **N2:** Nodal metastasis to periaortic, pericaval, superior mesenteric or celiac artery lymph nodes.

*Distant metastasis*

- **M0:** No distant metastasis
- **M1:** Distant metastasis.

Stage 0	Tis N0 M0
Stage I	T1 N0 M0
Stage II	T2 N0 M0
Stage III A	T3 N0 M0
Stage III B	T1-3 N1 M0
Stage IV A	T4 any N M0
Stage IV B	N2 disease M1 disease

**Nevin Staging** was also used in carcinoma gallbladder.

**Management**

Presentation	Indications	Management
Gallbladder polyp	Polyp > 1 cm, Age > 60 years with polyp Polyp with stones Documented increase in size	Open cholecystectomy
Incidental finding after cholecystectomy	T1a	No further management > port site excision
	T1b,2	Radical cholecystectomy and port site excision
	T3	Radical cholecystectomy and port site excision
	T4	Radical cholecystectomy and port site excision with extended right hepatectomy (IV - VIII)
Diagnosed case	T1	Open cholecystectomy
	T2	Radical cholecystectomy
	T3	Radical cholecystectomy
	T4	Radical cholecystectomy with extended right hepatectomy (IV - VIII)
Stage IV disease		Palliation of jaundice, gastric outlet obstruction and pain
Postoperative phase	Node positive T4	Adjuvant gemcitabine based chemotherapy

**Radical cholecystectomy** involves removal of gallbladder with segment IVB,V of liver with removal of pericyclic, pericholedochal and peripancreatic and interaortocaval lymph nodes.

- Debulking has no role in carcinoma gallbladder management
- Duodenal involvement is a contraindication to surgery as it is a marker of transcoelomic spread.

**Indications of CBD resection**

- Cystic duct margin positive
- Direct involvement of CBD
- Node positive disease if it interferes with nodal clearance.

**Indications of neoadjuvant chemotherapy in gallbladder cancer as of now**

- Only in trial setting
- Used in patients with unresectable nonmetastatic cancer.

**Indications of adjuvant chemotherapy**

- Node positive malignancy
- T4 tumors.

**Prognosis**

- Lymph node positivity
- Extramural extension especially into hepatoduodenal ligament
- Perineural invasion
- R0 resection.

**Q89. What are the risk factors for cholangiocarcinoma? Write the classification for cholangiocarcinoma.**

**Ans.**

**Risk factors for cholangiocarcinoma are as follows:**

- *Chlonorchis sinensis*, *Opisthorchis viverrini* infection
- Choledocholithiasis, cirrhosis
- Choledochal cyst
- Crohn's and ulcerative colitis
- Primary sclerosing cholangitis and recurrent pyogenic cholangitis
- Carrier of typhoid
- Biliary enteric anastomosis
- Dioxin, diabetes, obesity, oral contraceptive pills, smoking, thorotrast, isoniazid (**MN: DOSTI**)
- Radon, asbestos, nitrosamines, HIV, HBV, HCV.

**Bismuth-Corlette classification of cholangiocarcinoma**

- **Type I** - limited to the common hepatic duct, below the level of the confluence of the right and left hepatic ducts.
- **Type II**- involves the confluence of the right and left hepatic ducts
- **Type IIIa** - type II + extends to the bifurcation of the **right** hepatic duct
- **Type IIIb** - type II + extends to the bifurcation of the **left** hepatic duct
- **Type IV** - extending to the bifurcations of both right and left hepatic ducts OR multifocal involvement
- **Type V** - stricture at the junction of common bile duct and cystic duct

**Q. What is the management of cholangiocarcinoma.****Ans.**

- Patient presents with progressive obstructive jaundice and its associated symptoms
- Triple phase CT (MDCT) has 99% sensitivity and 75% specificity for diagnoses and is also important for vascular invasion
- If intrahepatic radicals are dilated or if there are equivocal CT findings, do MRCP
- Ca 19-9 – high preoperative levels are independent predictors of poor survival after attempted resection
- CT volumetry, ICG retention at 15 minutes and glucose/galactose tolerance tests are basic tests if liver resection is planned to assess remnant liver volume
- **Role of biopsy** – Clinically indeterminate origin, before starting palliative/experimental therapy
- **Preoperative biliary stenting** – No routine role  
Indications include the following:
  - Cholangitis, coagulopathy
  - Delay in surgery due to comorbidities
  - Before portal vein embolisation
- **Criteria of unresectability** are as follows:
  - Bilateral extension of tumor into secondary biliary radicals
  - Atrophy of one lobe with contralateral secondary biliary radical occlusion
  - Encasement or occlusion of main portal vein proximal to its bifurcation
  - Atrophy of one lobe with contralateral portal vein branch encasement or occlusion
  - One side secondary biliary radical and opposite side portal vein branch involvement (encasement or occlusion)
  - Periaortic, pericaval, SMA, celiac artery, peripancreatic, interaortocaval node involvement (N2)
  - Liver, lung or peritoneal metastasis.

**Management outline**

- **Papillary/well differentiated T1** – Extrahepatic bile duct excision
- **For all other resectable types, in Upper (Hilar)** – Bile duct resection with hepatectomy (caudate lobe always resected) and **in mid/lower CBD** – pancreaticoduodenectomy
- **Intraoperative frozen section** is done for interaortocaval lymph node and duct margin
- **Margin of resection** – Invasive/papillary/nodular – 2 cm macroscopic and 1 cm microscopic.

**Q90. Enumerate the risk factors for pancreatic cancer.****Ans. The risk factors for pancreatic cancer are as follows:**

- **Established** – Tobacco in any form, hereditary – FAMMM (Familial atypical multiple mole melanoma), Peutz Jegher syndrome, ataxia telangiectasia, hereditary pancreatitis, HNPCC, BRCA 2 mutation.
- **Associated** – Chronic pancreatitis, diabetes mellitus type 2, Obesity
- **Probable** – Physical inactivity, high carbohydrate diet, certain pesticides.

**Q. Write a note on management of a patient with pancreatic cancer.****Ans.**

- **Present as** asymptomatic mass, anorexia, weight loss, epigastric pain, obstructive jaundice or gastric outlet obstruction.
- **CECT with CT angiography is the imaging investigation of choice. Double duct sign is pathognomonic. (Duct penetrating sign is seen in chronic pancreatitis).**
- **Side viewing endoscopy and endoscopic ultrasound** is done when patient has a dilated main pancreatic duct with no mass lesion and/or no increase in tumor/marker or pancreatic enzyme level. It also helps in getting FNAC of the suspicious area when required.
- **Indications for preoperative biliary drainage are the same as in cholangiocarcinoma.**

**Resectability criteria are as follows:** (Encasement means  $> 180^\circ$  involvement of vessels and impingement/abutment means  $< 180^\circ$  involvement)

**Resectable** – No superior mesenteric vein or portal vein abutment/distortion/tumor thrombus/venous encasement with clear fat planes around hepatic artery, celiac axis and superior mesenteric artery.

**Unresectable** – Major venous thrombosis of superior mesenteric vein/portal vein for several centimetres or circumferential encasement of superior mesenteric artery, celiac axis or proximal hepatic artery or liver/peritoneal/distant lymph node/distant metastasis. All cases in between are called **Borderline resectable cases**.

**Management for all resectable and borderline resectable cases** in patients with good performance status is diagnostic laparoscopy laparotomy followed by whipple's pancreaticoduodenectomy or longmire traverse pylorus preserving pancreaticoduodenectomy. All resectable cases are followed by gemcitabine based adjuvant chemotherapy which improves survival in R0 resected carcinoma pancreas.

**Whipple procedure:** Resection of pylorus, duodenum, part of jejunum, CBD, gallbladder, pancreas head and uncinate process upto junction with SMV-PV followed by pancreaticojejunostomy, choledochojejunostomy and gastrojejunostomy in that order.

**Unresectable cases** are managed with FOLFIRINOX chemotherapy.

**Q91. Write a note on pseudomyxoma peritonei.****Ans.**

- It is also called Jelly belly
- Is a mucinous ascites most commonly originating from a perforated epithelial appendiceal neoplasm
- **Other tumors that can cause pseudomyxoma are as follows:**
  - Ovarian mucinous tumors [mucinous low malignant potential tumors (MLMP)]
  - Colorectal adenocarcinomas
  - Pancreas, gallbladder, fallopian tubes, uterine corpus and urachus, stomach, bile ducts are also implicated.

**Pathogenesis**

- Redistribution and distribution by gravity – motile tumor cells follow the flow along the peritoneal fluid direction and finally reach the open lymphatic lacunae on the

undersurface of the diaphragm as well as the lymphoid aggregates in the lesser and greater omentum. They also accumulate by gravity in pelvis, paracolic gutter, and retrohepatic space.

- Viscera which are peristaltic are spared due to their motility. The viscera that are relatively fixed are involved by the tumor.
- Abraded peritoneal surfaces due to previous surgery or multiple bowel adhesion sites are predisposed areas for the tumor cell deposition.

### **Clinical features**

- m.c. presentation – ovarian mass in female and acute appendicitis in male
- Median age – 55 years
- Female : male = 7:3
- Increasing abdominal distension
- Gastrointestinal obstruction
- Enterocutaneous fistula
- Internal enterovesical fistula
- New onset hernia.

### **Classification**

- **Ronnet classification**
  - Diffuse peritoneal adeno mucinosis (DPAM)
  - Intermediate group (IG)
  - Peritoneal mucinous carcinomatosis (PMCA)
- **Bradley modification**
  - High grade mucinous carcinoma peritonei
  - Low grade mucinous carcinoma peritonei – PMCA and IG are intermediate result group and DPAM is lowest grade

### **Investigations**

- CECT chest + abdomen + pelvis with IV and oral contrast
- Peritoneal carcinomatosis index (sugarbaker index)
  - Is a quantitative prognostic indicator
  - Used in both peritoneal carcinomatosis and pseudomyxoma peritonei
  - Abdomen and pelvis is divided into 9 regions and small bowel into 4 regions and lesion size goes from > 5 cm (3 points), 0.5-5 cm (2 points) and < 0.5 cm (1 point)
  - Maximum PCI – 39 suggest worst prognosis
- CEA, CA-19 9, CA-125 are tumor markers of prognostic value in this disease.

### **Management**

- Aims at complete removal of all the macroscopically visible tumor within the abdominal cavity
- Only the diseased peritoneum is removed. Also called peritonectomy of convenience or CRS.



**Surgery – Sugarbaker procedure**

Includes two main steps:

- **Maximal surgery** – lesser and greater omentectomy, right and left parietal peritonectomy, diaphragmatic and pelvic peritonectomy, liver capsulectomy, radical appendectomy, with or without right hemicolectomy and splenectomy
  - The aim of maximal surgery is to leave small tumor rests (< 2.5 mm) which are easily penetrated by the chemotherapy
- **Maximal chemotherapy** – given directly intraperitoneally
  - HIPEC – heated (40–44° C) intraperitoneal chemotherapy – heating is thought to improve drug distribution and penetration  
Drugs used in HIPEC are mitomycin c (10mg/m<sup>2</sup>), cisplatin, 5-Fluorouracil, oxaliplatin
  - EPIC – early postoperative chemotherapy (5-Fluorouracil)– not much supported.

**Prognosis**

Complete R0 resection – 5 year survival of nearly 85% is achieved.

**MISCELLANEOUS****Q92. Enumerate the factors responsible for the occurrence of burst abdomen.**

**Discuss the causes, clinical features and management of a patient with burst abdomen.**

**Ans.** It is also known as abdominal dehiscence. Literally, it means the abdomen rips open because of loss of the sutures that are holding the laparotomy wound together.

Predisposing factors are the same as those for incisional hernia and are rewritten here for recall purposes.

Patient factors	Operative factors
<b>Old age, male gender</b> <b>Malnutrition in perioperative period</b> Severe anemia, hypoproteinemia, vitamin A,C deficiency, zinc deficiency, advanced malignancy related cachexia, patients of longstanding dysphagia/gastric outlet obstruction <b>Obesity and sleep apnea</b> <b>Diabetes mellitus</b> <b>Chronic steroid use</b> <b>Jaundice</b> <b>Straining factor</b> Chronic constipation, chronic cough, bladder outlet obstruction, heavy weight lifting, ascites, prolonged Ileus	<b>Type of operation</b> <ul style="list-style-type: none"> <li>• Emergency surgeries for peritonitis, cancer, pancreatic surgeries, appendicular abscesses</li> </ul> <b>Incisions</b> <ul style="list-style-type: none"> <li>• Midline infraumbilical incision, Kocher's incision, McBurney's incision</li> </ul> <b>Technique</b> <ul style="list-style-type: none"> <li>• Mass closure</li> <li>• Nonanatomical closure</li> <li>• Suturing under tension</li> <li>• Wound infection or hematoma</li> <li>• Closure with absorbable sutures</li> <li>• Continuous suturing</li> <li>• Drains through the main wound</li> </ul>

**Clinical presentation**

- Presence of serosanguinous discharge from the operation wound on 4th or 5th day is an ominous sign that patient might have a burst abdomen
- If the patient already has a burst abdomen at presentation, then the bowel loops and omentum can be seen through the wound and confirms the diagnoses
- This finding is enough to diagnose a case of burst abdomen.

**Management**

- If the bowel loops are exposed, they should be covered with paraffin gauzes followed by moist towels to keep them from sticking to the towels and to keep them moist
- Patient's fluid electrolyte balance, analgesia, antibiotics must be taken care of and then patient is taken up for surgery
- During surgery, the bowel loops should be washed, peritoneal cavity irrigated and then the incision closed in layers meticulously with interrupted nonabsorbable sutures and anatomical closure
- If the abdomen is closed properly, a second dehiscence rarely occurs and it has been found in studies that the dehiscent abdomen has ironically developed strong scars.

The management outlined here is simple and routine. What is more intriguing is when the burst abdomen or rather the **dehiscence is planned one**, that is, the abdomen is not at all closed in an anatomical manner in the initial surgery.

This is **done in cases of damage control surgery** (discussed in the section on general surgery) wherein **during phase 3** (second look surgery after **phase 1** of damage control and **phase 2** of ICU resuscitation), in 35 to 45% of the patients, the patients develop abdominal compartment syndrome when closure is attempted (clinical pointer – whenever the abdominal wall closure causes the peak airway pressure to rise to > 10 cm of water, then the patient has abdominal compartment syndrome if the abdomen is still closed).

**An overview of management in these cases**

**Abdominal compartment syndrome** is defined as intraabdominal pressure greater than 20 mm Hg for a sustained period with or without abdominal perfusion pressure less than 60 mm Hg and new organ dysfunction or failure.

**Normal intra-abdominal pressure** - <8 mm Hg

Intra-abdominal hypertension - > or = 12 mm Hg

**Primary ACS** – Intra-abdominal cause

**Secondary ACS** – Extremity trauma with massive resuscitation or equivalent

**Recurrent ACS** – Repeated ACS [primary or secondary] despite treatment

In these cases of damage control, temporary abdominal closure devices which do not actually approximate the wound edges such as vacuum assisted closure device, vacuum pack, retention sutures, Bogota bag are used for just gaining time for further management. This allows an iatrogenic ventral incisional hernia (**phase 4**) to develop which is taken care of at a later date. In these cases, the difference from normal incisional hernia repair is that the endoscopic component separation or Rives–Stoppa repair alone has a very high recurrence rate of 22 to 33% and it needs to be supported by overlay or underlay biological mesh placement (**phase 5 or definitive closure**).

**Q93. Classify peritonitis and enumerate the clinical features of generalized peritonitis.**

**Ans.** Peritonitis is basically inflammation of the peritoneum.

**Types of peritonitis**

<b>Septic</b>	<b>Primary (Spontaneous bacterial peritonitis)</b>	<ul style="list-style-type: none"> <li>No intra-abdominal cause.</li> <li>Organism – <i>E.coli</i> in adults and <i>S.Pneumoniae</i> in children especially in children with nephritic syndrome or cirrhoses.</li> <li>Defined as &gt;250 polymorphs in ascetic fluid with a single organism in culture.</li> <li>If no organism is cultured, it is called neutrocytic ascites</li> </ul>
	<b>Secondary (Suppurative peritonitis)</b>	<ul style="list-style-type: none"> <li>Polymicrobial</li> <li>Causes such as peptic perforation (m.c.), intestinal perforation, etc.</li> </ul>
<b>Aseptic</b>	<b>Chemical peritonitis</b>	<ul style="list-style-type: none"> <li>Bile, urine, meconium, pancreatic fluid and gastric juice can all cause chemical peritonitis.</li> </ul>

**Present History**

- Localized abdominal pain which has now generalized
- Increased intensity of pain
- Exacerbation by movement, respiration, talking, coughing, touching, bending
- Relief on lying silent and still
- Vomiting and abdominal distension
- Breathlessness, palpitations, tachypnea, diaphoresis are important associated complaints.

**Past history**

- History of chronic illness such as tuberculosis, enteric fever, AIDS
- Chronic drug use – steroids, immunosuppressants, analgesic abuse
- Smoking, alcohol, malignancy, radiation.

**General physical examination**

- Agitated state or altered consciousness
- Patient prefer not to be moved or talked to and lie still
- Tachycardia, tachypnea, elevated temperature, signs of dehydration
- Anemia, lymphadenopathy may be present.

**Inspection findings**

- Decreased movement of abdomen with respiration
- Visible bowel loops, scars of previous surgery, hernia orifices
- Abdominal distension.

**Palpation**

- Diffuse tenderness, rebound tenderness, guarding and rigidity suggest peritonitis (always remember to rule out pancreatitis as a cause as it is a common surgical dictum)

“eat while u can, sleep while u can, operate when and what u can but, don’t mess with the pancreas”)

**Percussion and auscultation**

- Obliteration of liver dullness suggest perforation peritonitis
- Percussion tenderness
- Decreased or absent bowel sounds.

**Q94. Write a note on the management of a patient with mesenteric cyst.**

**Ans.**

- Mesentery is a reflection of the posterior peritoneum that connects the intestines to the posterior abdominal wall and carries blood vessel and nerves. The root of the mesentery extends from the ligament of treitz at the level of L2 and is approximately 6 inches long
- Mesenteric tumors can be **cystic or solid** with the cystic being more common (60%).

**Histologic classification of mesenteric cysts**

- Chylolymphatic cyst: endothelial lining (**m.c.**)
- Nonpancreatic pseudocyst has no lining, with a fibrous wall
- Mesothelial cyst: mesothelial lining
- Enteric duplication cyst: Enteric lining and double-muscle lining with neural elements
- Enteric cyst: Enteric lining (mucosa with no muscle layer)
- Urachal cyst from urachal remnant
- Chylolymphatic cyst arises because of congenital sequestration of lymphatics intra-abdominally which has no communication with the efferent lymphatic system
  - It most commonly arises in second decade of life and is more common in ileal mesentery
  - It is mostly unilocular, solitary, with thin walls and clear fluid content
  - It has an independent blood supply and therefore can be enucleated
- Enterogenous cyst arises from a diverticulum at the mesenteric border of the intestine and have only mucosal outpouching when it is called enteric cyst or it can form a duplication cyst of the intestinal lumen
  - It has a thicker wall and mucinous content
  - It has a blood supply common to the adjacent bowel and therefore, requires resection of the adjacent bowel during surgery.

**Clinical features**

- **Tillaux triad** gives the clinical features of mesenteric cyst which are – fluctuant swelling near umbilicus, movement perpendicular to the direction of the mesentery (**Tillaux sign**) and a zone of resonance around the cyst
- **Differentiated** from omental cyst because omental cyst is freely mobile in all directions (**Fothergill sign**) and from ovarian cyst because ovarian cyst can be pushed into pelvis while this cannot
- **Acute abdomen in mesenteric cyst can be because of torsion, hemorrhage, infection or rupture.**

**Investigation**

- CT is the investigation of choice.

**Treatment**

Surgery is the treatment of choice

Can be done by open method or laparoscopic method

- Enucleation for chylolymphatic cyst
- Resection anastomosis for enterogenous cyst
- Excision with removal of entire urachal remnant for urachal cyst
- Excision for mesothelial cyst.

# SECTION

# 3

## Genitourology

- Kidney and Ureter
- Urinary Bladder
- Prostate
- Testis and Scrotum
- Penis and Urethra
- Uro-oncology



## KIDNEY AND URETER

**Q1. Enumerate the types of renal calculi. Write its etiological factors.**

**Discuss the clinical features, complications and management outline of renal calculi.**

**Write a note on medical management of renal calculi.**

**Ans.** The types of renal calculi and its etiological factors are as follows:

<b>Calcium oxalate stones (m.c.)</b>	<ul style="list-style-type: none"> <li>• Hyperparathyroidism</li> <li>• Hypercalcemia</li> <li>• Short bowel syndrome</li> <li>• Hypercalcemia—other causes</li> <li>• Ethylene glycol poisoning</li> </ul>
<b>Struvite stones (TIPSSS stones – Triple phosphate stones, infection stones, phosphate stones, stag horn stones, silent stones, struvite stones)</b>	<ul style="list-style-type: none"> <li>• Need alkalinization with proteus infection</li> <li>• Urinary tract catheterization</li> <li>• Any cause of bladder outlet obstruction</li> </ul>
<b>Uric acid stones (m.c. radiolucent calculi)</b>	<ul style="list-style-type: none"> <li>• Myeloproliferative disorders</li> <li>• Gout</li> <li>• Lesh-Nyhan syndrome</li> </ul>
<b>Cystine stones</b>	<ul style="list-style-type: none"> <li>• Cystinuria</li> </ul>
<b>Xanthine stones</b>	<ul style="list-style-type: none"> <li>• Xanthinuria</li> </ul>
<b>Indinavir, triamterene stones, silicate stones</b>	<ul style="list-style-type: none"> <li>• Patients being treated with these medications</li> </ul>
<b>Other risk factors</b>	<ul style="list-style-type: none"> <li>• Immobilization</li> <li>• Vitamin A deficiency</li> <li>• Dehydration</li> <li>• Urinary stasis</li> </ul>

### Clinical features

- May be asymptomatic
- Seen in young males except staghorn calculi which are more common in females
- Pain in the flank region, associated with nausea, vomiting
- Associated urinary symptoms—increased frequency, hematuria, dysuria, burning micturition
- Pyonephrosis—high grade fever, pyuria, vomiting (sepsis)
- Renal failure—in cases of bilateral stone, or obstructing stone in a solitary functional kidney.

### Complications

- Hydronephrosis
- Pyonephrosis
- Renal failure
- Septicemia
- Non functional kidney—hypertension.



### Management outline

#### *Investigations*

- X-ray KUB (Kidney, Ureter, Bladder) – 90% stones are radio-opaque whereas 10% stones are radiolucent (**UXIT** - Uric acid, Xanthine, Indinavir, Triamterene)
- **Best investigation—NCCT**
- **USG**—to look for hydronephrosis
- **IVP** or contrast enhanced CT can be done to look for renal function if necessary before the surgery
- **Kidney function test** to rule out renal failure
- **Urine analysis**—
  - Urine routine examination, microscopy, culture and sensitivity
  - Alkaline urine predisposes to uric acid and struvite stones
  - Acidic urine predisposes to calcium oxalate stones, uric acid and cystine stones
- **Other useful investigations:**
  - **DMSA scan** for renal morphology
  - **DTPA scan** for renal function
  - **MAG-3** for renal perfusion study.

### Medical management

- Dietary changes
  - Increased oral fluid intake and decrease the animal protein intake (low purine intake) of the patient. Also decrease the fructose, sucrose and phosphate in the diet
  - Decrease the intake of fish, egg, meat for patients with cystinuria
  - Increase the intake of potassium, phytates and vitamin C in the diet of the patient
  - Spinach, asparagus and milk should be used with moderation
- Alteration of urinary pH so as to prevent crystallization of solutes: Alkalinization of urine in patients with cysteine/uric acid stones
- Drug therapy
  - Pain killers and antispasmodics for pain relief and antacids with prokinetics for relief of nausea and vomiting
  - Bendroflumethiazide for idiopathic hypercalciuria
  - Allopurinol and acetazolamide in uric acid stones
  - D-penicillamine in cystine stones
  - Antibiotics to treat urine infection and acetohydroxamic acid in struvite stones
  - Medical expulsive therapy—useful mostly for uretric stones: Drugs like calcium channel blockers, tamsulosin are given to relax uretric muscle and favour propulsion of uretric stone
- Treatment of metabolic cause and anatomic risk factors (bilateral vesicoUreteric junction obstruction/stricture/bladder outlet obstruction) is of utmost importance once the patient is relieved for the moment to prevent recurrence.

### Surgical management options

- ESWL (Extracorporeal shockwave lithotripsy)
- YAG laser lithotripsy (intracorporeal lithotripsy) with ureteroscopy

- Ureteroscopic stone removal
- Nephrolithotomy (incision through renal parenchyma)
- An—atrophic nephrolithotomy [Incision through brodel line (Avascular renal plane)]
- PCNL (Percutaneous nephrolithotomy)
- Pyelolithotomy (Incision through renal pelvis only)
- Extended pyelolithotomy (Incision in renal pelvis that extends into renal infundibulum and tip of calyces)
- Laparoscopic or open nephrectomy.

#### **Procedure preferences in renal calculi**

- Renal stone less than or = 2 cm: ESWL (Extracorporeal shockwave lithotripsy) but, if ESWL is contra/indicated then do PCNL (Percutaneous nephrolithotomy)
- Renal stone > 2cm, lower calyx stone, stones associated with distal obstruction, stones difficult to treat with ESWL (cysteine), staghorn stone- PCNL
- Stone in a nonfunctional kidney—laparoscopic/open nephrectomy.

### **Q2. Write a note on management of calculus anuria.**

#### **Discuss the management outline of bilateral renal or ureteric calculi.**

**Ans.** The occurrence of bilateral renal calculi or occurrence of unilateral renal calculi in a patient with single functioning kidney is one of the most common causes of calculous anuria.

#### **Management outline**

##### *Investigations*

- Ultrasound has very high sensitivity and specificity for diagnoses of hydronephrosis
- In cases with idiopathic retroperitoneal fibroses or metastatic ureteric obstruction, however, ultrasound can be false negative and MAG-3 and DTPA are better investigations in this case
- Bladder catheterization to diagnose anuria
- Rule out pre-renal and renal causes
- X-ray or NCCT
- Other investigations as mentioned above.

#### **Immediate management**

- **If the obstruction is ureteric (above urinary bladder),** Bilateral Double J stent placement through cystoscopy and if that fails bilateral percutaneous nephrostomy should be done to relieve the obstruction
- Once the obstruction is taken care of, evaluate the patient for surgery and plan the management as for single calculi as outlined in the question on single calculi management
- For the other causes of calculus anuria such as carcinoma bladder, rectum, uterus, ovary, the obstruction can also be **below urinary Bladder**, in which case, management is with Urethral or suprapubic catheterization.

#### **Difference in management outline for bilateral renal calculi includes the following:**

- The kidney with better function is treated first or the one that is having refractory pain or pyonephrosis should be treated first

- The recent concept is of bilateral simultaneous PCNL which is being done in selected cases with no distal obstruction, no complicated anatomy and by doing the difficult or symptomatic side first and doing the opposite side at the same time only if the first procedure does not have more blood loss and the patient is stable, fit for further anesthesia and acceptable time constraints.

**Metabolic workup in these patients is as follows:**

- Serum calcium level on 3 consecutive days
- Serum uric acid
- 24 hour urine examination for urine calcium, phosphate, urate and cystine
- Serum parathyroid and vitamin D level is done if calcium level shows abnormality.

**Rest medical and surgical management is as outlined above for single renal calculi.**

**Q3. Discuss the treatment options for management of stone in the lower third of ureter.**

**Ans.**

**Medical expulsive therapy**

- For stones < 10 mm in distal end of ureter.
- Tamsulosin 0.4 mg at night for six weeks is given.

**Ureteroscopic removal of stone**

- A cystoscope is inserted into the bladder via the urethra and guidewire is passed in the ureter
- The cystoscope is removed and uretroscope passed alongside the guidewire into the ureter
- The stone is visualized and either removed after grasping with a stone removing forcep or broken mechanically or with laser
- **Contraindication:**
  - Uncorrected coagulopathy
  - UTI
- **Advantages:**
  - Minimally invasive
  - No incision and its related complications
  - Day care surgery
  - Minimal trauma to ureter
- **Complications:**
  - Injury to ureter
  - Sepsis in cases of uncontrolled UTI.

**Laparoscopic ureterolithotomy**

- Can be intraperitoneal approach, or retroperitoneoscopically
- Advantages—minimally invasive, advantage in patients requiring other laparoscopic surgery like cholecystectomy in the same setting
- Disadvantages—trauma to ureter due to mobilization is more as compared to URS, longer hospital stay.

**Open ureterolithotomy**

- Not practiced now except in cases where an open surgery is contemplated for any other reason or in cases with failure of laparoscopic procedure.

**ESWL**

- Can also be used in ureteric stones. However, ureteroscopic removal is now the procedure of choice.

**Q4. Write a note on ESWL.**

**Ans.**

**ESWL:** Extracorporeal shockwave lithotripsy.

**Principle**

- Shockwaves are generated by a source external to the patient's body and are then propagated into the body and focused on a kidney stone using ultrasound, fluoroscopy or both.
- **Mechanism of lithotripsy:**
  - **Spallation:** Once the shockwave enters the stone, it will be reflected at sites of impedance mismatch. One such location is at the distal surface of the stone at the stone-urine interface. If the tensile wave exceeds the tensile strength of the stone, there is an induction of nucleation and growth of microcracks that eventually coalesce, resulting in stone fragmentation, which is termed spallation
  - **Squeezing-splitting or circumferential compression,** occurs because of the difference in sound speed between the stone and the surrounding fluid
  - **Cavitation**
  - **Shear stress** will be generated by shear waves that develop as the shockwave passes into the stone. The shear waves propagate through the stone and will result in regions of high shear stress inside the stone.

**Machine types**

- There are three primary types of shockwave generators: electrohydraulic (spark gap), electromagnetic, and piezoelectric
- In the electrohydraulic shockwave lithotripter, a spherically expanding shockwave is generated by an underwater spark discharge
- Electromagnetic lithotripter generates cylindrical waves that are focussed onto the target using an acoustic lens
- Piezoelectric generator also produces plane shockwaves with directly converging shock fronts.

**Indication**

- Renal stone  $\leq 2$  cm.

**Factors associated with high failure rate of ESWL**

- Staghorn stones, multiple stones, calcium oxalate monohydrate, cystine or brushite stones
- Stone in inferior calyx, stone associated with calyceal diverticulum, anatomic renal abnormality, distal obstruction

**Important considerations**

- ESWL with weaker shockwaves hurt less and the treatment can be given without general anesthesia
- To prevent infection, patient should be given prophylactic antibiotics
- Distal obstruction should be relieved by Double J stent placement before ESWL.

**Contraindications**

- **Absolute:** Pregnancy, coagulopathy
- **Relative:** Distal obstruction, severe renal failure, urinary infection
- **Other relative contraindications:** Uncontrolled hypertension, obesity, cardiac pacemaker in-situ.

**Complications**

- Steinstrasse as explained above
- Urinary infection
- Lung or kidney parenchymal injury
- Hematuria
- Cardiac ectopics.

**Q5. Write a note on PCNL.**

**Ans.** PCNL or percutaneous nephrolithotomy is a minimally invasive method for management of renal stones.

**Indications:**

- Staghorn calculi
- Renal stones >2 cm in size, stones associated with distal obstruction, stones difficult to break with ESWL-cystine stones
- Renal stones associated with PUJ obstruction.

**Contraindications:**

- Active UTI
- Uncorrected coagulopathy.

**Anesthesia**

- General anesthesia.

**Patient Position**

- Prone.

**Preoperative preparation**

- Urine culture should be sterile, preoperative antibiotics should be given.

**Procedure:**

- Patient is placed in lithotomy position and cystoscopy done to place ureteric catheter and a retrograde pyelogram is done to visualize the calyx
- Patient is then placed in prone position. Parts are cleaned and draped. Under fluoroscopy, the renal calyx containing stone is identified and site of puncture identified
- The desired calyx is punctured with a long needle (like spinal needle) and tract is dilated over a guide wire using graduated dilators

- Once tract is dilated, then the working sheath is placed and nephroscope is introduced to visualize the renal parenchyma
- The stone is identified and either removed or broken and then removed
- A nephrostomy tube is placed and working sheath is removed.

**Complications:**

- Haemorrhage
- Pleural effusion, pneumothorax—in case of supracostal puncture
- Injury to colon, duodenum and development of fistulas between urinary system and these organs
- Sepsis.

**Q6. Enumerate the indications of percutaneous nephrostomy.**

**Ans. Indications of percutaneous nephrostomy are as follows:**

- Pyonephrosis
- To allow recovery of renal function in a case of obstructing pelvic/ureteric stone
- To perform PCNL
- To give chemotherapy in postoperative cases of endoscopic resection of transitional cell carcinoma of pelvis
- In cases of ureteric stricture wherein a guidewire/ureteric catheter/stent cannot be placed via retrograde means
- To perform whitaker test to diagnose PUJ obstruction.

**Q7. Write a note on urinary diversion.**

**Ans.**

**Indications**

- After radical cystectomy
- After anterior, total or extended exenteration
- Retroperitoneal lesions with extrinsic compression
- Pediatric cases with bladder exstrophy or spinal cord dysraphisms or cloacal malformations
- Refractory neuropathic bladder
- Trauma cases with severe disruption of normal continuity
- Incurable urinary fistula or unresectable distal obstruction (Carcinoma rectum or prostate)
- Long ureteric strictures.

**Methods of urinary diversion**

<b>Intubations</b>	<ul style="list-style-type: none"> <li>• Percutaneous nephrostomy</li> <li>• Open nephrostomy</li> <li>• Ureteric Double J stent</li> <li>• Suprapubic cystostomy</li> </ul>
<b>Ureterocutaneous diversion</b> (Ureter open to exterior like stoma)	<ul style="list-style-type: none"> <li>• Cutaneous ureterostomy or pyelostomy</li> <li>• Transureteroureterostomy with end cutaneous ureterostomy</li> </ul>

*Contd...*

Contd...

<b>Ureterointestinal diversion</b> (Ureter open into intestine)	<ul style="list-style-type: none"> <li>• <b>Ureterosigmoidostomy</b> (Coffee) <ul style="list-style-type: none"> <li>– Leadbetter modification (Ureter open into sigmoid via a submucosal tunnel)</li> <li>– Transcolonic antireflux mechanism (Goodwin)</li> <li>– Detubularised sigmoid (Mainz principle – Detubularisation breaks the peristaltic wave and therefore protects from urine reflux)</li> </ul> </li> <li>• <b>Ileal conduit</b> – 15-20 cm terminal ileum starting 30 cm from IC junction is used in isoperistaltic fashion. Ureter open proximally, while the distal opening is ileostomy. Intestinal continuity is established by end-to-end anastomosis.</li> <li>• <b>Jejunal or colonic conduit</b> – More chances of hypokalemic metabolic acidoses.</li> <li>• <b>Continent diversion:</b> <ul style="list-style-type: none"> <li>– Method to provide patient with continence like natural urine control.</li> <li>– The continence can be through invaginated ileal loop (Kock's pouch) or appendix (Mitrofanoff procedure) or ileocecal valve (gilchrist/indiana/Mainz I or Stein pouch)</li> </ul> </li> </ul>
<b>Orthotopic neobladder:</b> Neobladder is constructed from intestinal loops and attached to the urethral remnant.	<ul style="list-style-type: none"> <li>• Mainz neobladder</li> <li>• Camey's enterocystoplasty</li> <li>• T-pouch ileal neobladder</li> <li>• Hautmann neobladder (largest reservoir capacity)</li> <li>• Studer neobladder.</li> </ul>

**Metabolic complications of intestinal urinary diversions are worth mention and are as follows:**

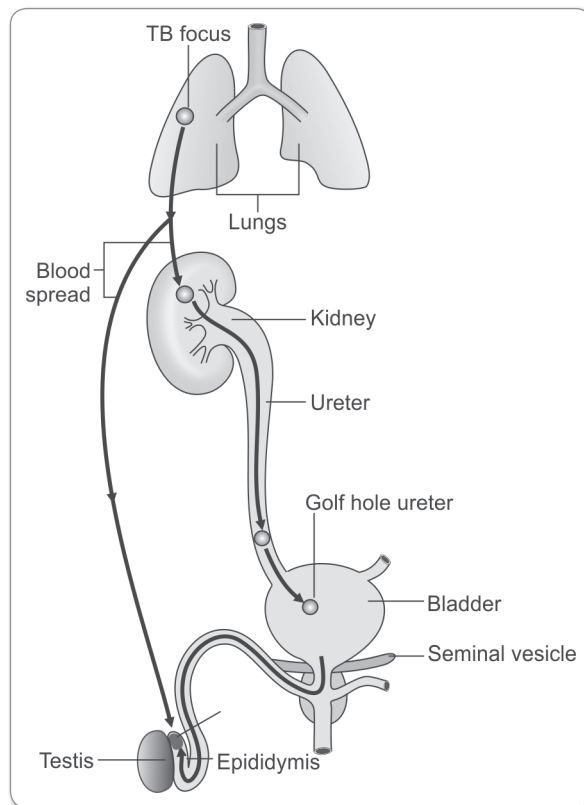
- **Loss of ileocecal valve and terminal ileum**
  - Vitamin B<sub>12</sub> deficiency
  - Hyperoxaluria and renal stones
  - Loss of conjugated bile acids and gall stones formation
  - Chronic diarrhea.
- **Ureterosigmoidostomy**
  - Increased reabsorption of urea and chloride and increased loss of potassium due to diarrhea as well as fluid loss
  - Increased bicarbonate loss due to loss of mucus of gut
  - This leads to hypokalemia, normal anion gap metabolic acidoses and hyperchloremia
  - Chronic bicarbonate loss results in bone demineralization and increased serum alkaline phosphatase.

**Q8. Write a note on etiology, clinical features and management of a patient with renal TB.**

**Ans. Etiopathogenesis**

- Genitourinary TB is caused by the spread of the mycobacteria through the bloodstream during the initial infection
- The kidney is usually the primary organ infected in urinary disease, and other parts of the urinary tract become involved by direct extension

- The initial infection occurs in the renal cortex, where the bacilli can remain dormant within the periglomerular cortical granulomas for decades. This dormant infection then becomes activated due to failure of the local immune response
- The organisms in the kidney settle in the blood vessels, usually those close to the glomeruli
- Caseating granulomas develop and consist of Langhans giant cells surrounded by lymphocytes and fibroblasts
- The healing process results in fibrous tissue and calcium salts being deposited, producing the calcified lesion, called as pseudocalculi
- The granulomas at the renal pyramids enlarge, form a tubercular abscess and if it keeps on enlarging, it can burst through the pelvicalyceal system and lead to pyuria—Sterile pyuria as no organisms are usually present in this urine
- Chronic pyuria leads to irritation, contraction at calyceal neck and PUJ leading to pyonephrosis
- The kidney becomes a bag of pus called putty kidney or cement kidney. There can be extensive calcification, which can lead to parenchymal destruction and eventual “autonephrectomy”. Extrarenal spread through rupture result in perinephric abscess.



**Fig. 1A:** Renal tuberculosis



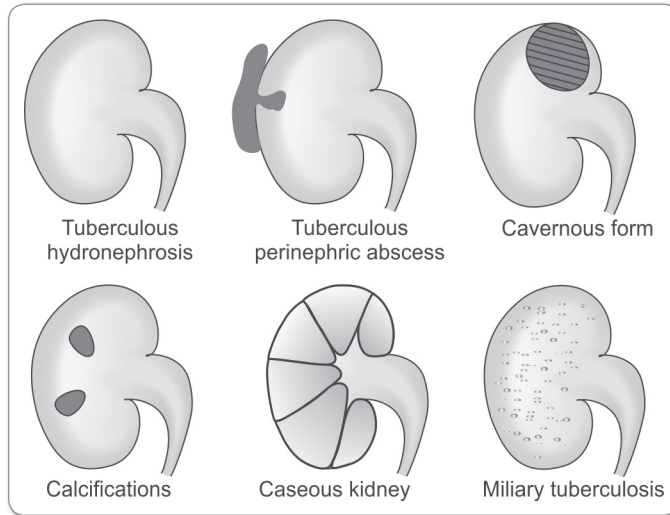


Fig. 1B: Renal tuberculosis

### Clinical features

- Painless increased frequency of micturition
- Hematuria
- Constitutional symptoms such as anorexia, fever with evening rise, weight loss
- Sterile pyuria.

### Involvement of the rest of genital tract

- Tuberculous ureteritis is always an extension of the disease from the kidney and leads to fibrosis and stricture formation. The site most commonly affected is the ureterovesical junction (UVJ)
- Chronic cystitis, tubercle formation in bladder mucosa, and finally Thimble bladder (small contracted bladder)
- Chronic prostatitis and seminal vesiculitis
- Testis is not involved
- Epididymal cysts and chronic draining scrotal sinus.

### Diagnosis

- **X-ray chest, mantoux test, total leukocyte count with lymphocyte count and ESR**
- **Urinalysis:** Sterile pyuria, microscopic hematuria
- **X-Ray KUB:** Calcification in kidney and genitourinary tract
- **IVP:** Investigation of choice in early renal TB. It helps to demonstrate infundibular stenosis, calyceal destruction (moth eaten calyx, caliectasis), ureteral stricture and hydronephrosis, renal or perinephric abscess, thimble bladder, function of kidneys
- **CECT:** Investigation of choice—helps in identifying calyceal abnormalities, hydronephrosis or hydroureter, autonephrectomy, amputated infundibulum, urinary tract calcifications, and renal parenchymal cavities. It also demonstrates function of the kidneys
- **USG:** Demonstrates hydronephrosis, caliectasis

- **RGP:** Demonstrates uretral stricture (pipe stem ureter) and helps in stenting them. Uretral catheterization helps in collecting urine sample for AFB identification and culture
- **Cystoscopy:** Golf hole ureter openings.

### Treatment

- **Multidrug anti tubercular treatment** is the mainstay of treatment according to the category of the patient and appropriate regimen
- Surgery is done after four to six weeks of ATT when indicated
- **The indications for nephrectomy are:**
  - A nonfunctioning kidney with or without calcification
  - **Extensive disease involving the whole kidney, together with hypertension and UPJ obstruction and**
  - **Coexisting renal carcinoma**
- **For PUJ obstruction,** pyeloplasty
- **For UVJ obstruction,** excision of diseased segment of ureter together with uretric reimplantation is done
- **Renal abscess** persisting after two months of ATT and increasing in size on ATT can be aspirated by percutaneous route.

### Q9. Write a note on renal trauma management.

**Ans.** Renal trauma can be blunt or penetrating.

**American association for the surgery of trauma organ injury severity scale for the kidney is as follows (AAST grading):**

Grade	Type	Description
I	Contusion	Microscopic or gross hematuria with normal urologic studies
	Hematoma	Subcapsular, nonexpanding without parenchymal laceration
II	Hematoma	Nonexpanding perirenal hematoma confined to renal retroperitoneum
	Laceration	<1 cm parenchymal depth of renal cortex, no urinary extravasation
III	Laceration	>1 cm parenchymal depth of renal cortex, no collecting system rupture or urinary extravasation
IV	Laceration	Parenchymal laceration extending through renal cortex, medulla, and collecting system
	Vascular	Main renal artery or vein injury with contained hemorrhage
V	Laceration	Completely shattered kidney
	Vascular	Avulsion of renal hilum, devascularizing the kidney

### Clinical features

- Haematuria in a patient with polytrauma may be indicator of renal trauma
- Degree of hematuria and severity of renal injury do not always correlate
- Shock and hemodynamic instability.

### Indications for imaging:

- All penetrating trauma patients with suspected renal injury
- All blunt trauma patients with gross hematuria

- All patients with microscopic hematuria and shock (systolic blood pressure <90 mmHg any time during evaluation and resuscitation)
- Blunt abdominal trauma with rapid deceleration injuries.

**Imaging options:**

- CECT abdomen with CT urography
- On table single shot IVP to stage the injured kidney and assess the function of the uninjured kidney intra-operatively.

**Management:***Non-operative management*

- The isolated renal injury, without significant associated injuries, occurs more commonly from blunt trauma and in most circumstances can be managed **nonoperatively**. The exception is major grade V vascular pedicle avulsion injuries
- Isolated renal injuries with parenchymal lacerations and even segmental arterial injury can have active bleeding well controlled by **angiographic embolization**
- Patients with grade IV parenchymal lacerations who have well-contained hematomas can be observed expectantly
- They should be closely monitored for bleeding, with vital signs, serial hematocrit readings, and pulse rates. If urinary extravasation is present, serial renal CT scanning should be instituted
- If significant urinary extravasation persists beyond 48 hours, placement of an internal ureteral stent for drainage often prevents prolongation of the extravasation and decreases the chance of perirenal urinoma formation
- If nonoperative management is selected for a patient with gross hematuria whose injury has been well staged with appropriate imaging, hospital admission and bed rest are required. Once the gross hematuria clears, ambulation is allowed; should gross hematuria recur, bed rest is reinstated. Ambulation without any sequelae allows hospital discharge with close clinical follow-up.

*Operative management*

- **Absolute indications**
  - Evidence of persistent renal bleeding
  - Expanding perirenal hematoma or pulsatile perirenal hematoma.
- **Relative indications**
  - Urinary extravasation
  - Nonviable tissue more than 20%
  - Delayed diagnosis of arterial injury/ segmental arterial injury
  - Incomplete staging
  - Surgery being done for hemodynamic instability due to other associated abdominal injuries.
- **Renal Exploration**
  - It is best done by a transabdominal approach
  - The renal vessels are isolated before exploration to provide the immediate capability to occlude them if massive bleeding should ensue when Gerota's fascia is opened

- The kidney is exposed by incising the peritoneum lateral to the colon, followed by mobilization of Gerota's fascia. This maneuver often requires release of the splenic (left) or hepatic (right) attachments of the colon
- Gerota's fascia is then opened and the kidney with injury is completely dissected from the surrounding hematoma.
- **Renal reconstruction**
  - This is done following debridement of nonviable tissue, hemostasis by individual suture ligation of bleeding vessels, watertight closure of the collecting system, and coverage or approximation of the parenchymal defect
  - Polar renal injuries can be managed with reconstruction or partial nephrectomy. The open parenchyma should then be covered when possible by a pedicle flap of omentum, absorbable mesh, peritoneal graft, or retroperitoneal fat.
- **Renovascular injuries**
  - Vascular repair requires occlusion of the involved vessel with vascular clamps. The perforated main renal vessels injured by penetrating trauma can be repaired with 5-0 nonabsorbable vascular suture.
- **Indications for nephrectomy**
  - Total nephrectomy would be indicated immediately in extensive renal injuries when the patient's life would be threatened by attempted renal repair.

**Q10. Write a note on the management of ureter injury and enumerate the options of ureteric reconstruction procedures.**

**Ans.**

**American association for the surgery of trauma organ injury severity scale for the ureter (AAST grading):**

Grade	Type	Description
I	Hematoma	Contusion or hematoma without devascularization
II	Laceration	< 50% transection
III	Laceration	≥ 50% transection
IV	Laceration	Complete transection with < 2 cm devascularization
V	Laceration	Avulsion with > 2 cm devascularization

**Types of ureteric injury:**

- **External injury:** Penetrating injury, missile injuries
- **Surgical injury:** Hysterectomy, colorectal surgery, pelvic ovarian tumour surgeries, abdominal urethropexy, abdominal vascular surgery
- **Deliberate ureteric transaction:** Radical cystectomy surgery, dismembered pyeloplasty surgery, after ureterocele excision, pelviureteric junction obstruction surgery, retrocaval ureter surgery
- **Ureteroscopic injury:** Avulsion of ureter during dormia basketting or ureteroscopic removal of a renal or ureteric stone

**Diagnosis:**

- **Intraoperative suspicion:** Due to the anatomic location of surgery, trajectory of missile.
- **Delayed presentation:** Fever, peritoneal irritation, leucocytosis.
- **Imaging:** IVP findings are often subtle and nonspecific, for example, delayed function, ureteral dilation, and ureteral deviation. **CECT with CT urography** may detect urinary extravasation on delayed images to suggest ureteral injuries. **Retrograde pyelography** is used to detect and grade injuries seen on the IVP or CECT.

**Management:**

- **Timing:** Except for patients with delayed identification or iatrogenic injury identified postoperatively, all other patients should undergo immediate repair.
- **Principles of repair**
  - The repair should be tension free
  - Mucosa to mucosa, end to end, spatulated anastomosis
  - Full thickness single layer anastomosis
  - Water-tight anastomosis with internal stenting
  - Always retroperitonealise repair or isolate it by omentum wrap or interposition
- **Preferred procedures**
  - **Upper ureteral injuries:** Ureteroureterostomy, auto transplantation of kidney, bowel interposition (Monti procedure)
  - **Mid ureteral injuries:** Ureteroureterostomy, transureteroureterostomy
  - **Lower ureteral injuries:** Ureteric reimplantation by Lich-Gregoir or Leadbetter Politano technique, ureteroneocystostomy, psoas bladder hitch, boari flap.
- **Delayed recognition:** Ureteric stenting retrograde via cystoscopy or nephrostomy and antegrade stenting to divert urine to allow local inflammation to heal followed by delayed repair of injury after four to six weeks.
- **Final management:** All these management are stented anastomosis, which are evaluated after 6 weeks by a RGU and if the RGU is normal, then the stent is removed and patient followed up with ultrasound or lasix renogram. If RGU is not normal, patient is managed with restenting or as per the RGU findings.

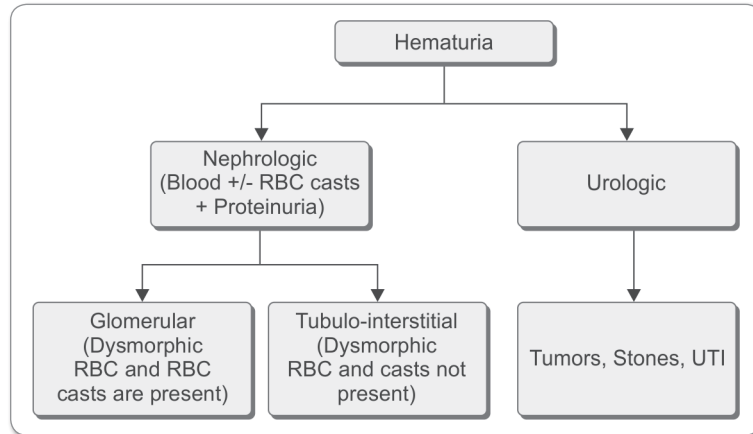
**Q11. Enumerate the causes of hematuria. Discuss the management outline of a case of posttraumatic hematuria.**

**Ans.** Hematuria is passage of blood in urine and according to definition, > 3 RBC/Hpf suggest hematuria.

**Types and differentiation**

- The hematuria is identified by presence of RBC in urine
- If RBC's are not present, then it can be hemoglobinuria or myoglobinuria
- In hemoglobinuria, serum is stained. Whereas, in myoglobinuria, serum is clear as in normal patients
- Once the patient is confirmed to have hematuria, then proceed to microscopy examination and estimation of protein level to classify it amongst nephrologic and urologic causes

- Nephrologic causes are further glomerular or tubular as shown in the flow chart below:



#### Glomerular causes

- IgA nephropathy [m.c.]
- Membranous, membranoproliferative, mesangioproliferative glomerulonephritis
- Lupus nephritis
- Subacute bacterial endocarditis
- Focal segmental glomerulonephritis.

#### Tubule-interstitial causes

- Papillary necrosis which can be due to diabetes, sickle cell disease or analgesic nephropathy
- Renal artery or venous thrombosis
- Disseminated intravascular coagulation
- Thrombophilia.

#### Urologic causes

- Renal trauma
- Tumor
- Stone disease
- Instrumentation.

#### Other

- Essential hematuria: No proteinuria, no casts, normal RBC's
- Non-glomerular hematuria of parenchymal origin
- Exercise induced hematuria: Seen in long distance runners at the conclusion of run. It is usually the presenting feature of glomerular disease such as IgA nephropathy.

#### Basic investigations to be done in evaluation of any patient with hematuria

- Cystoscopy
- Urine cytology
- IVP/CT urography/ultrasound

**Further management will depend on findings of these tests.**

Management of a trauma patient with hematuria is outlined in the question on renal trauma.

**Q12. Enumerate the types of pyelonephritis and discuss its management.**

**Ans.** Renal infections are a very serious type of infection. They can be classified as follows:

<b>Pyelonephritis</b>	Acute	Emphysematous (Triad of fever, flank pain and vomiting)
	Chronic	Xanthogranulomatous pyelonephritis
	Special conditions	Acute form in pregnancy/children/ patients with urinary retention
<b>Renal abscess and carbuncle</b>	Seen in diabetes and immunosuppressed	
<b>Perinephric abscess</b>	Mode of spread	Hematogenous Periureteral lymphatics Renal cortical abscess Appendicular abscess
<b>Pyonephroses</b>	Causes	Acute pyelonephritis Infected hydronephrosis Calculus (m.c. cause)
<b>Renal tuberculosis</b>		

- **Pyelonephritis** is defined as inflammation of the kidney and renal pelvis
- It can be acute form or chronic form.

**Clinical presentation**

- Abrupt onset of fever with chills
- Unilateral or bilateral flank or costovertebral angle pain and/or tenderness
- Dysuria, increased urinary frequency, and urgency
- It may present with acute renal failure
- On examination, patient may be febrile, hypotension may be present
- Costovertebral angle tenderness may be present.

**Diagnosis**

- Hemogram will show neutrophilic leucocytosis
- C-reactive protein may be elevated and kidney function test may be deranged
- Urinalysis usually reveals numerous WBCs, often in clumps, and bacterial rods or chains of cocci. Leukocytes may be present
- The presence of large amounts of granular or leukocyte casts in the urinary sediment is suggestive of acute pyelonephritis
- A specific type of urinary cast characterized by the presence of bacteria in its matrix has been demonstrated in the urine of patients who had acute pyelonephritis
- Urine culture: May yield *E coli*
- Blood cultures may also grow gram negative organisms
- IVP: Generalized renal enlargement more than 1.5 cm as compared to opposite side, focal renal enlargement, delayed pyelogram, diminished nephrogram, renal scarring
- Renal USG: Demonstrates oedema, enlargement

- CECT abdomen with urography: Useful in patients who do not respond to treatment after 48 hours.

**Management**

- **If no features of urosepsis is present:** Oral fluoroquinolones for 7–10 days
- **If patient has features of sepsis,** inpatient treatment for 14-21 days is given with parenteral antibiotics- Ampicillin plus aminoglycoside/ fluoroquinolones/third generation cephalosporins. **Supportive therapy** with antipyretics, and IV fluid to maintain hydration
- **Follow up:** Repeat urine cultures should be performed on the fifth to the seventh day of therapy and 10 to 14 days and 4 to 6 weeks after discontinuing antimicrobial therapy
- **Patients who relapse** usually are cured by a second 14-day course of therapy, but occasionally a 6-week course is necessary.

**Acute focal nephritis:**

- Acute renal infection in which a heavy leukocyte infiltrate is confined to a single renal lobe or multiple lobes
- The radiological investigations reveal a mass lesion suggestive of renal abscess or tumor.

**Treatment**

- Hydration
- Intravenous antimicrobial agents for at least 7 days, followed by 7 days of oral antimicrobial therapy.

**Emphysematous pyelonephritis:**

- It is an acute necrotizing parenchymal and perirenal infection caused by gas-forming organisms
- Classic triad of fever, vomiting, and flank pain
- Common in diabetics
- E. coli is the most commonly identified organism.

**Investigation:** X-Ray KUB demonstrates mottled gas in renal area.

- **CT** is the imaging procedure of choice in defining the extent of the emphysematous process and guiding management.

**Management:**

- Emphysematous pyelonephritis is a surgical emergency
- Most patients are septic, and fluid resuscitation and broad-spectrum antimicrobial therapy are essential
- If the kidney is functioning, medical therapy can be considered
- Nephrectomy is recommended for patients who do not improve after a few days of therapy. If patient improves, percutaneous nephrostomy may be done to relieve any obstruction.

**Xanthogranulomatous pyelonephritis**

- Xanthogranulomatous pyelonephritis is a chronic form of pyelonephritis seen in patients with urolithiasis especially staghorn calculus with proteus infection



- It usually occurs unilaterally and presents with fever, flank pain, palpable flank mass.
- CT is the investigation of choice and urinalysis will reveal leucocytes, bacteria and protein
- Management is with partial or total nephrectomy alongwith appropriate antibiotics.

**Q13. Enumerate the causes of pyonephrosis and discuss its management.**

**Ans.** Pyonephrosis is accumulation of purulent material in kidneys which lead to loss of functioning renal parenchyma and deterioration of renal function.

- **Causes**
  - Renal calculus disease (m.c.)
  - Infection in hydronephrosis
  - Progression of acute pyelonephritis.
- Kidney here becomes a multilocular cavity full of pus or purulent urine and gradually becomes non-functional
- **Clinical features**
  - **Triad** = flank mass + anemia + fever
  - Fever may be associated with chills and rigors
  - Tracking down of pus into urinary bladder may result in cystitis.
- **Investigations**
  - Ultrasound abdomen will show floating contents with increased echoes suggestive of pus
  - X-ray KUB may reveal a calculus
  - DTPA scan will show renal function.
- **Treatment**
  - Immediate initiation of antibiotics is the first step
  - After that, the obstructed system is to be drained using percutaneous or open nephrostomy > double J stent placement and removal of the obstruction (stone or other cause of hydronephrosis if present)
  - If the kidney is completely non-functional, nephrectomy is warranted.

**Q14. Write a note on hydronephrosis and discuss its management.**

**Ans.** Hydronephrosis is aseptic dilatation of kidneys due to obstruction.

**Causes**

Unilateral	Bilateral
<b>Intraluminal</b> <ul style="list-style-type: none"> <li>• Calculus disease</li> <li>• Sloughed papilla in papillary necrosis [Diabetes, analgesic abuse, sickle cell disease (<b>DAS</b>)]</li> </ul> <b>Intramural</b> <ul style="list-style-type: none"> <li>• Neoplasm</li> <li>• Inflammatory</li> <li>• Ureterocele</li> <li>• Congenital stenosis</li> </ul>	Bilateral hydronephrosis will occur when the bladder outlet or urethra is obstructed as seen in the following causes: <b>Congenital</b> <ul style="list-style-type: none"> <li>• Urethral atresia</li> <li>• Posterior urethral valve</li> </ul> <b>Acquired</b> <ul style="list-style-type: none"> <li>• All causes of bladder outlet obstruction such as benign prostatic hypertrophy, carcinoma prostate, urethral stricture and phimoses</li> </ul>

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Unilateral	Bilateral
<b>Extramural</b> <ul style="list-style-type: none"> <li>• Infiltration from adjacent structures: Cervix, prostate, rectum, cecum and colon</li> <li>• Ormond disease</li> <li>• Retrocaval ureter</li> </ul>	<ul style="list-style-type: none"> <li>• Retroperitoneal fibrosis</li> </ul>

**Clinical features**

- Pain in the flank region
- **Dietl's crises:** Pain and swelling in loin region that disappears after passing urine is Dietl's crises
- Flank swelling
- Bilateral variety present with features of bladder outlet obstruction
- Can lead to chronic renal failure
- Infection in hydronephrosis leads to pyonephrosis.

**Investigations**

- Ultrasound will show hydronephrosis, calculi, pyonephrosis if present
- Prenatal ultrasound will detect hydronephrosis which help in deciding the role of fetal surgery in the case
- IVP or DTPA scan is done for functional evaluation
- Older test: **Whitaker test** is seldom performed now.

**Treatment***Indications of surgery*

- Increasing hydronephrosis
- Infection in hydronephrosis
- Severe pain
- Renal parenchymal loss and diminishing function.

*Options*

- Follow up with analgesics and ultrasound is limited to mild cases only
- Double J stenting and percutaneous nephrostomy are both useful in relieving the obstruction
- Double J stent is preferred unless there is pyonephrosis wherein, a percutaneous or open nephrostomy is the preferred method
- Treatment of the underlying cause is the definitive management once the obstruction is relieved.

**Q15. Write a note on Wilms' tumor.****Ans.**

- Wilm's tumor is also known as nephroblastoma
- It is the most common primary renal malignancy in the age of 2 to 5 years and is overall 2nd most common after neuroblastoma in abdomen

**Syndromic associations**

- **Beckwith-Wiedemann syndrome**
  - Hemi-hypertrophy, macroglossia, midline abdominal wall defects such as exomphalos/divarication of recti or umbilical hernia, ear defects and neonatal hypoglycemia
  - Wilm's tumor, renal medullary cysts and other genitourinary abnormalities are common
  - Defective gene is on **chromosome 11**.
- **WAGR syndrome or WAGRO syndrome:**
  - Wilms tumor, aniridia, genitourinary abnormalities and gonadoblastoma, mental retardation, obesity in childhood
  - Defect is on **chromosome 11**.
- **Denys – Drash syndrome**
  - Characterized by Wilm's tumor, gonadal dysgenesis and nephropathy
  - Typical triad is: Pseudohermaphroditism, mesangial sclerosis of kidney and Wilm's tumor
  - Defect is on **chromosome 11**
- **Perlman syndrome**
  - Macrosomia, macrocephaly, visceromegaly
  - Renal hamartomas and Wilm's tumors
  - Defect is on **chromosome 2**.

**Pathology**

It contains elements of all the three germ layers:

- Epithelium
- Stroma (muscle, cartilage, fat, bone)
- Blastema.

**Clinical features**

- Flank mass
- Flank pain
- Hematuria
- Hypertension.

**Investigations**

- CT is the investigation of choice for staging of Wilm's tumor.

**Staging**

This can be **prechemotherapy** [As used in America developed as national Wilm's tumor staging group (NWTSG)] wherein, staging is done at surgery and adjuvant chemoradiation is used or **postchemotherapy** staging [International pediatric oncology society in Europe (SIOP staging)] wherein neoadjuvant chemotherapy is given irrespective of histology and then postchemotherapy surgery and staging is done.

**Treatment**

- Surgical excision followed by chemoradiation is the preferred course of treatment especially for stage I and II patients

- Stage III and IV are considered for preoperative chemotherapy followed by surgery
- Stage V is treated with induction chemotherapy and if favourable response then surgery
- Chemotherapy: Vincristine, doxorubicin, etoposide and cyclophosphamide (VDEC) or Vincristine and actinomycin combination regime
- Radiation is also used to treat the pulmonary metastasis associated with Wilm's tumor.

**Q16. Write a note on horse shoe kidney.**

**Ans.** Write the development and embryological basis of horse shoe kidney as described in the question on development of urinary system.

**Add the following points.**

- It is also known as super kidney
- It is the most common fusion abnormality. Other is crossed fused ectopia which is less common
- The horse shoe kidney is not simply fusion of kidneys. It has the following abnormalities in development:
  - **Fusion** of the lower poles
  - **Nonascent** to normal position because of the inferior mesenteric artery blocking the ascent
  - **Nonrotation** as the fusion occurs before the anteromedial rotation. So, the axes is vertical and calyces posteriorly directed whereas renal pelvis is anteriorly directed
  - **Ureter is anteriorly placed** and therefore, more prone to obstruction by compression and infection.

**Clinical features**

- It is more common in males
- Incidence is 1:400
- Most are asymptomatic
- Can present with abdominal pain
- **Rovsing's sign:** Hyperextension of spine produces abdominal pain, nausea and vomiting. (**Always remember:** There are two rovsing sign. One is this and the other is Rovsing's sign in appendicitis. Also remember the Rovsing's operation that is used in Polycystic kidney cases.)

**Investigations**

- Usually picked up on ultrasound or CT done for finding the cause of abdominal pain
- IVP is the investigation of choice. It shows the above developmental abnormalities. It also gives **flower vase like appearance** of ureter and **hand-shaking sign** of kidneys
- Angiography is done to rule out aberrancy in renal vessels so as to prevent complications in surgery.

**Associated abnormalities**

- Vesicoureteral reflux
- Pelviureteric junction obstruction
- It is also associated with trisomy 18 and turner syndrome
- Anorectal malformations, hypospadias and undescended testis are other associated anomalies.

**Treatment**

- Usually management is not to be done as most patients are asymptomatic
- In symptomatic cases treatment of choice is pyeloplasty. Care is taken to keep the isthmus fused as it is. The procedure can be performed open or laparoscopically.

**Q17. What is PUJ obstruction? Discuss its management.**

**Ans.** Pelviureteric junction obstruction is obstruction at the junction of renal pelvis and ureter that causes block in the normal urinary flow.

**Causes**

- Congenital or acquired causes
- Congenital PUJ obstruction is the most common cause of antenatal hydronephrosis
- **Congenital:**
  - Urothelial ureteral fold
  - Aberrant renal vessel or absent smooth muscle in the segment resulting in an aperistaltic segment
  - Anomalous collagen collar that fails to recanalise during the development phase
  - Ischemic scarring during development.
- **Acquired**
  - Renal trauma
  - Upper tract transitional cell carcinoma at that site
  - Renal infection with scarring
  - Renal calculus disease
  - Instrumentation: Double J stent, percutaneous or open nephrostomy, pyelolithotomy.

**Associations**

- PUJ obstruction of the opposite kidney [m.c.]
- Vesicoureteric reflux
- Horse shoe kidney.

**Clinical presentation**

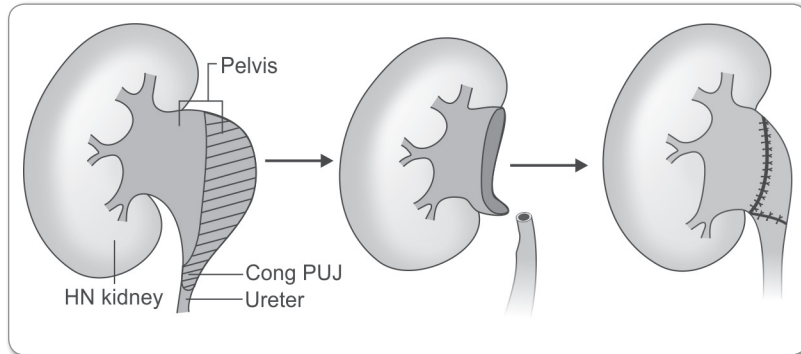
- Antenatal detection on ultrasound
- Asymptomatic
- Palpable abdominal lump.

**Investigations**

- Ultrasound shows a dilated renal pelvis with a collapsed proximal ureter
- IVP was previously the investigation of choice but, now has been replaced by MAG3 scan
- CT: Shows hydronephrosis, caliectasis and collapsed ureters
- DTPA is not found to be as useful nowadays because DTPA depends purely on glomerular filtration and in obstructive uropathy, glomerular function declines earlier and more rapidly than does tubular function. Therefore, DTPA can be used only if the patient has good renal function
- MAG3 labeled with technetium is therefore the agent of choice
- Diuretic (Furosemide) renogram is also used sometimes to differentiate between obstructing and non-obstructing hydronephrosis
- Test of historical importance: Whitaker test.

**Treatment**

- Asymptomatic uncomplicated cases: Observation
- Symptomatic/Complicated cases
- **Open or laparoscopic anderson hynes dismembered pyeloplasty** is the gold standard procedure. Here, the redundant portion of pelvis, involved segment of ureter are cut and then spatulated, stented, tension free single layer anastomosis is done to restore continuity
- **Open or laparoscopic modified Anderson hynes dismembered pyeloplasty.**



**Fig. 2:** Anderson hynes pyeloplasty

- **Foley's V-Y plasty**
- **Pyeloplasty using spiral flaps**
- **Ureterocalicostomy** in cases with failure of pyeloplasty, lower caliectasis with small pelvis or in patients with exaggerated intrarenal pelvis
- **Endoscopic pyelotomy or laser pyelotomy** can be done in patients with small pelvis or intra-renal pelvis with the involved segment smaller than 2 cm in maximum length
- Patients with non-functioning kidney or functioning kidney but severe refractory and recurrent infections, intractable pain and refractory hypertension are candidates for **nephrectomy**.

**Q18. Enumerate the differential diagnoses of renal lump in an adult patient.**

**Enumerate the investigations and classification of renal lumps.**

**Ans.** Renal masses can be broadly divided into pediatric and adult masses depending on the age of presentation.

**Pediatric renal masses**

- Wilms tumor (unilateral or bilateral form)
- Nephroblastomatosis
- Renal cell carcinoma
- Mesoblastic nephroma (most common renal tumor in neonates)
- Multicellular cystic renal tumor
  - Cystic nephroma
  - Cystic partially differentiated nephroblastoma

- Clear cell sarcoma
- Rhabdoid tumor
- Angiomyolipoma
- Renal medullary carcinoma (in sickle cell trait patients)
- Metanephric adenoma
- Lymphoma
  - Hodgkin
  - Non-Hodgkin.

#### **Adult renal masses**

- Renal cell carcinoma
- Renal lymphoma
- Angiomyolipoma
- Oncocytoma
- Renal medullary carcinoma
- Renal sarcoma
- Metastasis.

#### **Non neoplastic enlarged mass like kidneys**

- ADPKD
- ARPKD
- Hydronephrotic enlarged kidney
- PUJ obstruction
- Pyonephrosis.

#### **Suspicious renal mass**

- **Ultrasound**
  - Hyperechoic solid mass
- **Intravenous pyelogram**
  - Increased tissue density
  - Irregular outline
  - Mass effect with calcification
  - Septation
- **CT** for some other reason
  - Increased enhancement of solid mass
  - Complex cyst

Whenever a patient presents with any renal mass having above characteristics, then that patient should be worked up with a dedicated renal CT/MRI (Both have complications as CT contrast can cause contrast nephropathy whereas MR contrast gadolinium can cause nephrogenic systemic fibroses).

#### **CT classification of renal masses (Bosniak)**

I	No septa, No calcification and No enhancement	No risk of malignancy
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II	No enhancement and Fine septation or calcification (Hair like)	No risk of malignancy
IIF	No enhancement and Hyperdense lesion < or = 3 cm Multiple fine septa/calcification Nonenhancing thick calcification Nonenhancing hyperdense lesion > 3 cm	3-5%
III	Measurable enhancement Measurably thick septa Irregular wall	Upto 50% risk
IV	Enhancing soft tissue components Thick septa Irregular wall	80-90% risk

## URINARY BLADDER

**Q19. Enumerate the types and discuss the management of bladder calculi.**

**Ans. Types of bladder calculi**

<b>Migrant calculi</b>	<ul style="list-style-type: none"> <li>Formed in the upper tracts and pass into the bladder</li> <li>They are retained there owing to their larger size or associated bladder neck obstruction</li> </ul>
<b>Primary/ idiopathic calculi</b>	<ul style="list-style-type: none"> <li>Stones in the bladder in the absence of obstruction, local disease, neurologic lesion, or known primary infection</li> <li>Dietary phosphate deficiency as in human breast milk in endemic areas, Vitamin A, B1, B6 deficiency, Magnesium deficiency are predisposing factors</li> <li>These can be ammonium acid urate (uric acid stones are most common form in adults overall whereas ammonium urate stones are the most common primary bladder stones) with/out calcium oxalate</li> </ul>
<b>Secondary bladder calculi</b>	Most often related to predisposing factors such as <ul style="list-style-type: none"> <li>Urinary stasis</li> <li>Recurrent urinary tract infection due to bladder outlet obstruction</li> <li>Neurogenic bladder dysfunction</li> </ul>

### Clinical features

- Asymptomatic
- Pain during micturition
- Terminal hematuria
- Strangury
- UTI
- Breaking of the urinary stream.

### Diagnosis:

- Cystoscopy is the modality of choice
- Ultrasonography
- X-ray pelvis and KUB – Most are radiolucent urate stones.



**Treatment:**

- **Open cystolithotomy:** A lower abdominal incision is given and opened in layers. Urinary bladder is identified and peritoneum swept upwards. Stay sutures over bladder are taken and bladder opened. Stone is removed and bladder closed in double layers with or without putting a cystostomy catheter. The incision is then closed in layers
- **Cystoscopic litholapaxy:** Stone is removed via stone grasping forceps after crushing it
- **Cystoscopic lithotripsy:** Larger stones are broken with electrohydraulic lithotripsy and removed.

**Q20. Differentiate between extraperitoneal and intraperitoneal bladder injury.****Write a note on clinical features and management of bladder injury.**

**Ans.** Bladder injuries occur mostly after a blow to the full bladder and are associated with pelvic fracture.

**Clinical features:**

- Most commonly associated with history of blunt abdominal injury
- **Classic triad:** Suprapubic pain, Inability to pass urine, hematuria
- Associated pelvic fracture in 90% cases.
- **Abdominal examination:**
  - Tenderness in lower abdomen without a palpable bladder in extraperitoneal injury
  - Diffuse tenderness and abdominal distension and ileus in intraperitoneal injury
  - Signs of external injury like bruise
  - Positive pelvic compression test in associated pelvic fracture.

**Investigations:**

- **Ultrasonography abdomen:** Collapsed bladder, perivesical collection, ascites in intraperitoneal bladder injury, rent in bladder may be detected
- **Cystography:** Gold standard investigation to detect bladder rupture. It shows distorted bladder with perivesical contrast extravasation with streaks of contrast going into fascial planes giving rise to **sun burst appearance**  
The pattern of contrast extravasation in extraperitoneal injury is termed as **flame sign**.
- **CT cystogram:** Useful as it can be clubbed with CT abdomen for trauma.

**Types of injury**

Extraperitoneal bladder injury	Intraperitoneal bladder injury
More common 80%	Less common
Injury to bladder below the peritoneal reflection	Injury above peritoneal reflection
Pelvic fracture is the commonest cause	Blow or fall on full bladder is the common cause
Present with the triad of bladder injury as mentioned above	Present with signs of peritonism or ileus
Radiology	
• Flame sign or pear sign in cystogram	• Ground glass appearance on X-ray

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Extraperitoneal bladder injury	Intraperitoneal bladder injury
Treatment	
<ul style="list-style-type: none"> <li>Simple catheter drainage in patients not requiring surgery for any other cause</li> <li>Surgical repair done if patient is undergoing laparotomy for other reason or in patients with bladder neck injury or protruding bone fragments into bladder.</li> </ul>	<ul style="list-style-type: none"> <li>Always repair surgically with laparotomy with suprapubic cystostomy with bladder repair.</li> </ul>

### Management of bladder injury

- **Extraperitoneal injury:**
  - Simple catheter drainage of bladder for two weeks. Prior to catheter removal, a dye study is performed to see for absence of contrast extravasation and ensure bladder healing.
  - **Indications of surgery in extraperitoneal injury:**
    - Patient requiring surgery for other associated injury of abdomen
    - Pelvic bone fracture fragment projecting into bladder
    - Associated rectal injury to prevent rectovesical fistula
    - Bladder rent extending onto bladder neck
    - Pelvic fracture requiring open reduction
    - Open pelvic fracture
    - Associated vaginal injury
    - Inadequate bladder drainage or clots in urine
    - Penetrating bladder injury
- **Intraperitoneal bladder injury:**
  - Treated by laparotomy and repair of bladder injury in two layers
  - The bladder is drained by putting an intra operative suprapubic catheter or per urethral catheter
  - A retropubic drain is also put.

### Q21. Write a note on vesicovaginal fistula.

**Ans.** Most common acquired fistula of the urinary tract

#### Causes

- **Most common causes**
  - **Developed countries:** Injury to the bladder at the time of gynaecologic (hysterectomy, Cesarean section, forceps delivery), urologic, or other pelvic surgery such as anterior colporrhaphy or cystocele repair, anti-incontinence surgery.
  - **Developing countries:** Prolonged obstructed labour due to cephalopelvic disproportion with resulting pressure necrosis of the anterior vaginal wall, bladder, bladder neck, and proximal urethra from the baby.
- **Other common causes**
  - Pelvic fracture
  - Endoscopic bladder procedures such as biopsy

- Advanced pelvic malignancy
- Radiation to pelvic organs.

### Clinical features

- Constant urine drainage per vagina
- Pelvic examination with a speculum helps in assessing the fistula, its margins, any growth and local infection
- The presence of a VVF may be confirmed by instillation of a vital blue dye (methylene blue or indigo carmine) into the bladder per urethra and vaginal packing and observing for discolored vaginal drainage. Staining at the distal end of the packing suggests urinary incontinence or a urethrovaginal fistula, whereas proximal staining suggests a VVF
- A ureterovaginal fistula should be excluded with a clean vaginal packing, the intravenous administration of indigo carmine (or other vital dye), and a repeated pad test. Blue staining at the proximal end of the pad after this maneuver suggests a ureterovaginal fistula.

### Investigation

- Cystoscopy is mandatory
- Cystography or voiding cystourethrography and an upper tract study should be performed in patients being evaluated for a VVF.

### Treatment

#### • Timing of surgery

Cause is obstructed labour	3-6 month delay
Cause is radiation induced fistulas	6-12 months delay
Cause is surgical trauma induced urinary tract fistula	Immediate

#### • Route of surgery: Transvaginal or transabdominal

Transvaginal	Transabdominal
Vaginal incision	Abdominal incision
<b>Indications</b> <ul style="list-style-type: none"> <li>• Uncomplicated low fistula</li> </ul>	<b>Indications</b> <ul style="list-style-type: none"> <li>• Large fistulas</li> <li>• Location high in a deep narrow vagina</li> <li>• Small-capacity bladder requiring augmentation</li> <li>• Radiation fistulas</li> <li>• Failed transvaginal approach</li> <li>• Need for ureteral reimplantation</li> </ul>
<b>Timing of repair:</b> Can be done immediately in the absence of infection	Usually done after an interval of 3-6 months
It is the favoured approach for posthysterectomy vesicovaginal fistulas	
<b>Adjunctive flap options:</b> Labial pad flap, gluteal skin or gracilis myocutaneous flap	<b>Adjunctive flap options:</b> Omental, peritoneal, rectus abdominis flap

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Transvaginal	Transabdominal
<b>Advantages</b> <ul style="list-style-type: none"> <li>• Short operative time</li> <li>• Less blood loss</li> <li>• Less postoperative morbidity</li> <li>• Quick convalescence</li> </ul>	<b>Advantages</b> <ul style="list-style-type: none"> <li>• High fistulas in deeper vagina are easily approached</li> <li>• Useful in cases where transvaginal route fails</li> </ul>
<b>Disadvantages</b> <ul style="list-style-type: none"> <li>• Fistulas high on the vaginal cuff are difficult to repair.</li> <li>• Risk of vaginal shortening is present</li> </ul>	<b>Disadvantages</b> <ul style="list-style-type: none"> <li>• Fistula low on trigone or near the bladder neck are difficult to repair</li> <li>• Risk of vaginal shortening is absent</li> </ul>

**Q22. Discuss the pathway of micturition reflex and write a note on neurogenic bladder.**

**Ans. Nerves of continence**

- Sympathetic—T11 to L2 (Inferior hypogastric plexus to hypogastric nerve to pelvic plexus)
- Parasympathetic—S2 to S4 gives branches to pelvic plexus. Supplies bladder and internal urethral sphincter
- Motor—S2 to S4. Supplies external urethral sphincter.

**Neural control of micturition and urine storage**

Storage phase	Voiding phase
Low vesical afferent activity	High vesical afferent activity on filling leads to stimulation of supraspinal and spinal reflexes
External sphincter remains contracted (Somatic motor control)	External sphincter relaxes because the pudendal nerve is inhibited by supraspinal reflexes
Inhibition of parasympathetic outflow and therefore no detrusor contraction.	Stimulation of the parasympathetic pathway leads to urethral smooth muscle relaxation and detrusor contraction
Internal urethral sphincter is in tonic state of contraction when sympathetic is stronger than parasympathetic	Inhibition of sympathetic pathway leads to internal urethral sphincter relaxation
Active sympathetic outflow results in detrusor inhibition and tonic internal sphincter contraction	The result is detrusor contraction and internal and external sphincter relaxation
Ganglionic inhibition via spinal reflexes also play a role	Both spinal and supraspinal reflexes play a role here

**Important pathways**

- **Sacral loop** or local loop
- **Supraspinal sensory afferents** to brain stem center at dorsal pontine tegmentum (Barrington center)
- **Cortico regulatory afferents** from the spinal centres as well as Barrington nucleus and efferents control the responses through all these centres to the sacral loop
- **Prefrontal cortex** also sends in fibres and aids in social control of micturition pathway

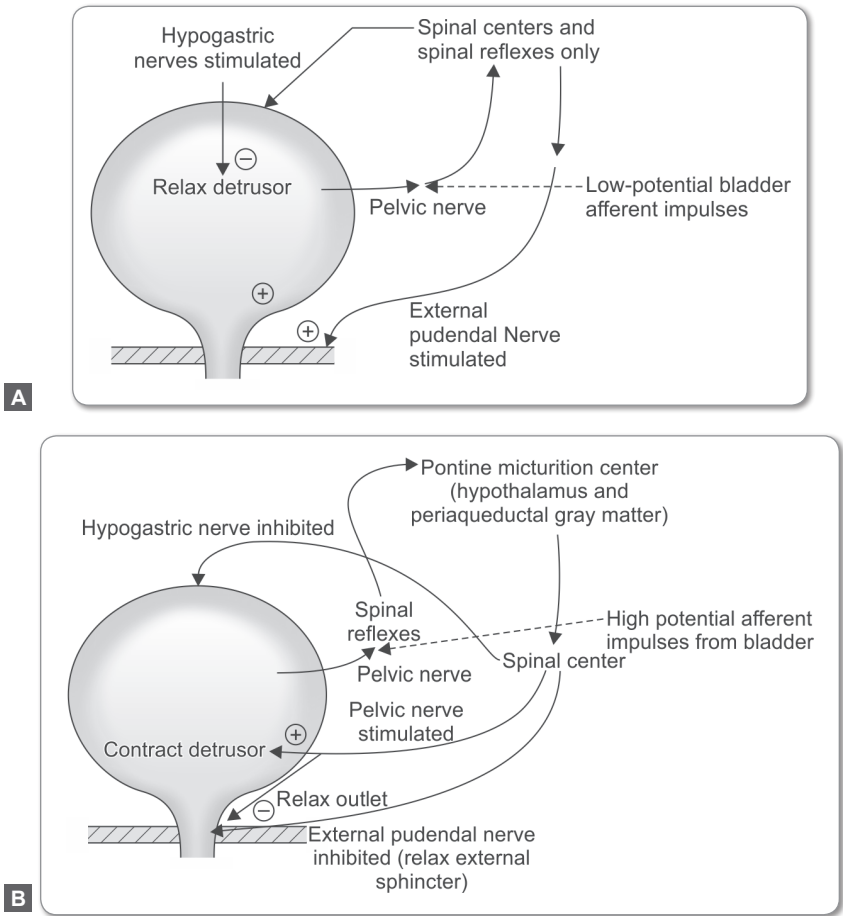


Fig. 3: Micturition reflex pathway; A. Guarding reflex for urine storage; B. Micturition voiding reflex

**Neurogenic bladder**

- This is basically a bladder with defect in the above mentioned nerve connections, therefore called a neurogenic bladder
- Its types are as follows:

**Lapides classification**

<b>Sensory</b>	Diabetes, Pernicious anemia, Tabes dorsalis
<b>Motor/Parasympathetic</b>	Pelvic surgery, trauma
<b>Uninhibited bladder</b>	The corticoregulatory pathway to the sacral micturition center is lost. So the Efferent control is lost. This results in <b>urge incontinence, frequency and urgency</b> because though the efferent supraspinal control is lost, the sensory afferents are intact and produce these symptoms.

Contd...

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<b>Reflex neurogenic bladder</b>	All suprasacral controls - both motor and sensory controls are lost and leads to <b>dyssynergia and insensate incontinence</b>
<b>Autonomous bladder</b>	Even the sacral control is lost. This leads to <b>overflow incontinence</b> .

### Hald-Bradley classification

Anatomical classification of neurogenic bladder is as follows:

<b>1</b>	Defect in pathway from cortex to pontine micturition center
<b>2</b>	Suprasacral fibres
<b>3</b>	Sacral fibres to urinary bladder
<b>4A</b>	Defect in the suprasacral control of Pudendal nerve
<b>4B</b>	Defect in the afferent feedback control pathway from detrusor and periurethral striated muscle to pudendal nerve

The management of these cases begins with urodynamic studies. The management of the neurogenic bladder is basically the management of the different types of incontinence that occur in it as discussed in the question on incontinence and control of the cause of neurogenic bladder.

### Q23. Enumerate the types of incontinence and discuss its management.

Ans.

- Incontinence is not being able to control the micturition pathway
- Incidence is about 5% in men and 15 to 20% in women. The incidence increases with age
- Important types and causes of incontinence are as follows:

<b>Continous incontinence</b>	<ul style="list-style-type: none"> <li>• Vesicovaginal fistula</li> <li>• Vesicourethral fistula</li> <li>• Female ureteral duplication</li> </ul>
<b>Stress incontinence</b>	<ul style="list-style-type: none"> <li>• Seen mostly in multiparous women</li> <li>• It occurs usually due to a weak sphincter mechanism</li> <li>• Incontinence occurs in the times of increased intra-abdominal pressure such as coughing, straining, valsava</li> </ul>
<b>Urge incontinence</b>	<ul style="list-style-type: none"> <li>• It is also known as uninhibited neurogenic bladder</li> <li>• Here, the corticoregulatory motor efferent pathways to sacral micturition center are lost so that there is involuntary incontinence from even a low volume and low pressure bladder</li> </ul>
<b>Overflow incontinence and chronic retention</b>	<ul style="list-style-type: none"> <li>• It is an autonomous neurogenic bladder</li> <li>• Loop 3 of Hald and Bradley classification</li> <li>• All sacral connections to the bladder are lost</li> <li>• This leads to large residual volumes at low pressure</li> <li>• This is also caused by long standing bladder outlet obstruction</li> </ul>
<b>Other causes of incontinence</b>	<ul style="list-style-type: none"> <li>• Sensory/motor/reflex neurogenic bladder all can cause incontinence</li> </ul>

**Management**

- History and physical examination
- Serum creatinine, kidney function tests
- Ultrasound KUB  
IVP
- **Urodynamic studies includes:**
  - **Uroflowmetry** (flow rate vs flow time graph. Normal is bell shaped curve. Flattened curve suggest obstructive uropathy whereas broken or interrupted curve suggests impaired bladder contractility/Use of abdominal straining for voiding)
  - Postvoid residual volume
  - Electromyogram of periurethral striated muscles
  - Cystometrogram (detrusor muscle)
  - Urethral pressure profiling.

**Treatment options**

- **Stress urinary incontinence**—Pelvic floor exercises, open colposuspension, transvaginal tape procedure
- **Decreased bladder compliance**
  - **Augmentation “Clam” enterocystoplasty** (Bowel is attached to bladder neck to create new bladder upto bladder neck) OR
  - **Substitution enterocystoplasty** (Near total supratrigonal cystectomy with detubularised ileocecal bladder anastomosis to trigone)
- **CISC** – Clean intermittent self-catheterization
- Maintenance of local hygiene
- Bladder training
- Medical management—Botulinium toxin/ Anti-cholinergic drugs to inhibit bladder activity/ Adrenergic blockers to decrease the bladder neck strength.

**Q24. Write a note on chyluria.**

**Ans.** It is presence of lymphatic fluid in urine.

**Causes**

- Genital filariases
  - Occurs in young adults with/ out microfilaremia and occurs before genital elephantiasis.
  - Dying worms provoke a lymphatic obstruction with proximal lymphangiolar dilatation
  - Rupture of a lymph varix into the collecting system leads to chyluria.
- Schistosomiasis
- Tuberculosis
- Idiopathic lymphorenal fistula
- Pregnancy
- Thoracic duct obstruction
- Mesenteric adenitis
- Metanephric adenoma
- Prior surgery

It results in hypoalbuminemia and anasarca

It is usually intermittent and remits spontaneously with bed rest.

### Management

- Urinalysis reveals milky white urine and eosinophilia
- Localization is done using cystoscopy/retrograde pyelogram/CT or MR urography and lymphangiogram.
- Treatment
  - Most cases resolve spontaneously
  - For filarial chyluria, treat with DEC and low fat diet
  - Retrograde insillation of silver nitrate or povidone iodine as sclerosing agents into the collecting system is the first line treatment
  - **Surgery – laparoscopic or open tranperitoneal or retroperitoneal nephrolysis** wherein, complete mobilization of kidney is done with skeletonisation of renal hilar vessels and upper ureter followed by ligation of the lymphatic channels and omental wrapping around the hilum.

### Q25. Discuss the clinical features and management of vesicoureteric reflux.

#### Ans.

- It is the abnormal flow of urine from bladder into ureter and kidneys
- Vesico-ureteric reflux is the most common congenital anomaly of the genitourinary tract
- It is Autosomnal dominant inheritance. Hardikar syndrome is a rare hereditary cause of vesicoureteric reflux
- It is associated with posterior urethral valve in 50% cases and epispadias in 40% cases
- Most cases are asymptomatic and resolve spontaneously.

#### Types

- **Primary**—the longitudinal muscle of intravesical part of the ureter is defective or the intravesical part is short in length thereby allowing reflux.
- **Secondary**—caused due to other causes such as neurogenic bladder, posterior urethral valve, urethral or meatal stenosis and epispadias all of which cause increased intravesical pressure and reflux.

#### Presentation

- Asymptomatic
- Prenatal hydronephrosis
- Recurrent urinary tract infections
- In secondary variety, the cause may be the presentation as in epispadias.

#### Complications

- Occurs due to reflux of urine and can result in pyelonephritis, renal scarring and chronic renal failure, renovascular hypertension and uremia.

#### Investigations

- MCU is the investigation of choice
  - Grading on MCU is as follows:



<b>I</b>	Reflux present, but ureter is nondilated
<b>II</b>	Reflux upto calyces but, the system is nondilated
<b>III</b>	Minimal to mild dilatation of ureter, pelvis and calyces with normal fornices
<b>IV</b>	Blunting of fornices
<b>V</b>	Gross dilatation with tortuous ureter.

- Urine analysis, microscopy and culture
- DMSA for morphology of kidney, renal scarring, pyelonephritis
- DTPA for renal function in advanced disease
- Abdominal ultrasound to rule out hydronephrosis, pyelonephritis.

### Treatment

*Medical management is preferred in grade I to III as well as in grade IV unilateral disease.*

- Co-trimoxazole is the drug of choice and is prescribed for prolonged durations. It is not to be used in children less than 6 weeks age. In these cases, amoxicillin is the drug of choice.
- Nitrofurantoin, nalidixic acid, cephalosporins can also be used
- Routine urine analysis and urine culture is to be done at follow up every 3 months.

*Surgical management is done in following situations*

- All patients with bilateral grade IV or Any Grade V reflux
- Recurrent urinary tract infections and recurrent renal scarring with progressive deterioration in renal function
- All patients with secondary VUR that does not resolve on its own after the correction of the primary cause
- Persistent reflux in patients nearing puberty.

*Surgical options*

- Ureteric reimplantation procedure (Open/Laparoscopic)
  - Lich-Gregoir technique which is direct implantation of ureter
  - Cohen procedure
  - Leadbetter Politano technique—implantation followed by submucosal anti-reflux tunnel
- Endoscopic sting procedure—subureteric transurethral injection of teflon paste.

### Q26. Write a note on ectopia vesicae.

**Ans.**

- Ectopia vesicae is also known as bladder exstrophy
- It is a defect in the anterior abdominal wall in its infraumbilical portion.

### Etiopathogenesis

- It occurs due to failed development of the cloacal membrane and underlying mesoderm.
- It is a part of exstrophy—epispadias complex and involves outward exposure of the urinary bladder through a defect in the infraumbilical abdominal wall
- Males are more commonly involved
- Hereditary predisposition is present.

### Features include

- A defect in the abdominal wall

- Exstrophied bladder as well as a portion of the urethra
- External rotation of the pelvis
- Anterior displacement of anus and rectal prolapse
- Separation of the pubic symphysis
- Shortening of the pubic rami
- Chordee and epispadias
- Undescended testis and inguinal hernia in males
- Females frequently have a displaced and narrowed vaginal orifice, a bifid clitoris, and divergent labia.

#### **Prenatal diagnosis:**

- Lower abdominal bulge
- Nonvisualization of urinary bladder on prenatal ultrasound
- Widening of iliac crests

#### **Complications**

- Skin complications due to urinary incontinence and soiling.
- Risk of malignancy (urinary bladder adenocarcinoma)
- Recurrent urinary tract infections
- Hydronephrosis.

#### **Treatment**

- **Primary closure**
  - In patients with an appropriately sized and functioning bladder (Elasticity, contractions and capacity)
  - Absolute contraindications to primary repair
    - Bladder abnormality
    - Bilateral hydronephrosis
- **Staged procedure**
  - Urinary bladder, posterior urethral and abdominal wall closure is performed first.
  - Enterocystoplasty (Augmentation or substitution) is used to increase bladder capacity
  - If the bladder is completely fibrosed and useless, then a cystectomy and urinary diversion is performed
  - Penile lengthening, bladder neck reconstruction are performed at second stage
  - The surgery for epispadias occurs at 6 to 12 months of age.

## **PROSTATE**

#### **Always Remember**

**Zones of prostate are given by McNeal and are as follows:**

<b>Anterior</b>	Fibromuscular zone anterior to central zone	
<b>Central</b>	Zone between transitional and anterior zone	
<b>Transition</b>	Zone surrounding the urethra	Prone for BPH
<b>Peripheral</b>	Posterior and on sides of central and transition zone	Prone for malignancy

Prostatic capsules are as follows:

<b>Capsule of peripheral zone</b>	Removed in BPH
<b>Anatomical capsule</b>	Periprostatic venous plexus lies between anatomical capsule and periprostatic sheath
<b>Periprostatic sheath</b>	Is derivative of endopelvic fascia and is contiguous with Denonvillier fascia

**Q27. Enumerate LUTS. Discuss the management of a patient with benign prostatic hypertrophy.**

**Discuss the medical management of a case of benign prostatic hypertrophy.  
and**

**Q28. Discuss TURP and its complications.**

**Ans.**

- The incidence of BPH is increasing along with age from 15–20% at 45 years age to nearly 90% at 80 years age and above
- Benign prostatic hypertrophy affects the transition zone of McNeal prostatic zones and is a spectrum of disorder with variety of manifestations as follows:
  - Lower urinary tract symptoms (**LUTS**)
  - Bladder outflow obstruction (**BOO**) (Urodynamic finding only)
  - LUTS + BOO
  - **Complicated BPH**—Acute retention (Usually the first symptom)/chronic retention/bladder stone/UTI/renal insufficiency/bilateral hydronephrosis

#### **LUTS : Lower urinary tract symptoms**

These can be classified as:

- **Symptoms related to voiding:**
  - Hesitancy
  - Poor flow (unimproved by straining)
  - Intermittent stream
  - Post-void dribbling
  - Sensation of incomplete bladder emptying
  - Urinary retention
  - Double voiding (repeat voiding within 2 hours)
- **Symptoms related to storage:**
  - Frequency
  - Urgency and urge incontinence
  - Nocturia and nocturnal enuresis
  - Dysuria.

#### **Diagnostic evaluation**

- **International prostate symptom score (IPSS)**
  - Mild—1-7
  - Moderate—8-19
  - Severe—20-35

- **Abdominal examination** for lumps or palpable bladder
- **Urodynamic study** to rule out bladder outlet obstruction
- **Serum creatinine** levels
- **Urine examination** for microscopy and culture-sensitivity
- **PSA level, Transrectal ultrasound** if indicated. Always remember that DRE findings do not correlate with the symptoms
- **Upper urinary tract imaging and cystourethroscopy** if indicated.

### Bladder outlet obstruction (On urodynamics)

*Causes include*

- BPH, bladder neck stenosis, prostate cancer or urethral stricture
- Detrusor instability or overactivity or hypertrophy (Marion disease)
- Neurogenic bladder (Diabetes, Alzheimer, Parkinson's disease, Stroke)

*Urodynamic values (for voided volume > 200 ml/minute)*

- $Q_{max} < 10$  ml/seconds
- Voiding pressure > 80 cm H<sub>2</sub>O
- Chronic retention (Postvoid residual volume > 250 ml).

### Management of BPH:

- **Conservative management:** If patient doesn't have bothersome symptoms.
- **Medical management:**

<b>Alpha blockers</b>	<ul style="list-style-type: none"> <li>• Relax the <b>smooth muscle</b> in prostate and bladder neck</li> <li>• Decreases the resistance to urine flow in urethra</li> <li>• Drugs include Prazosin, Terazosin, Tamsulosin etc.</li> <li>• Cause early symptom relief but little effect on disease progression if used alone</li> <li>• Always to be started for early symptom relief</li> </ul>
<b>5-alpha reductase inhibitors</b>	<ul style="list-style-type: none"> <li>• Block the conversion of testosterone to dihydrotestosterone</li> <li>• Helps in diminishing the androgenic stimulus for prostate growth especially affects the <b>epithelial proliferation</b> of prostate</li> <li>• Drugs include Dutasteride, Finasteride etc.</li> <li>• Cause improvement in symptoms but after a long term of use</li> <li>• It also decreases the incidence of future episodes of urinary retention</li> <li>• Nearly 4 to 6 months therapy is required to have its maximum benefit</li> <li>• % reduction in prostate size is of around 15 to 20% at 6 months</li> </ul>
<b>Combination therapy</b>	<ul style="list-style-type: none"> <li>• Has the best response</li> <li>• It causes increase in maximal flow rate by 2 to 3 ml/second and IPSS improvement of 30 to 60%</li> <li>• Prostate size reduction of 50 to 60% is also achieved by combination therapy</li> </ul>

- **Surgical management:**
  - **TURP (trans urethral resection of prostate)**
    - Gold standard for the surgical management of BPH
  - **Indications :**
    - Bothersome symptoms not relieved on medical management
    - Recurrent UTI and hematuria due to BPH
    - Azotemia

- Refractory urinary retention
- Second episode of urinary retention
- Secondary vesical calculi.
- **Contraindications:**
  - Uncorrected coagulopathy
  - Active urinary tract infection
  - Unable to make lithotomy due to hip ankylosis
- **Pre operative investigations:**
  - Urine analysis
  - IPSS
  - Serum PSA levels
  - DRE
  - Cystoscopy is not routinely done, used only if helpful to exclude other causes of the symptoms or help plan the type of surgery.
- **Anesthesia:** GA/SA/Perineal block
- **Position:** Dorsal lithotomy
- **Preoperative antibiotics:** A first-generation cephalosporin or a combination of this with gentamicin. It is valuable to maintain the patient on oral antibiotics until the catheter is removed.
- **Surgical technique**
  - Preliminary endoscopy and urethral calibration.
  - Bladder is filled with approximately 150 ml of a nonhemolytic irrigation solution (e.g., 1.5% glycine). 1.5% glycine is the irrigating solution of choice with osmolarity of 200 mosm, flow of 300 ml/min and height of pint < 60 cm.
  - The resection **begins at the bladder neck**, starting at the 12 o'clock position and carried down to the 9 o'clock position in a stepwise fashion
  - The adenoma is resected down to the level where the apparent circular fibers of the bladder neck become visible
  - If, at the completion of the entire resection, the bladder neck appears to be partially obstructing, bladder neck incision at the 6 o'clock position is done to prevent bladder neck contracture
  - Care must be taken not to resect too deeply in the region of the posterior aspect of the vesical neck to prevent undermining of the trigone
  - The adenoma is removed immediately proximal to the external sphincter mechanism, **preserving the verumontanum** to avoid sphincter injury.
- **Postoperative care:** Patient are catheterized and returning urine should be light pink in color.
- **Complications:**
  - Bleeding
  - Urinary retention
  - Capsular perforation
  - Bladder perforation
  - Retrograde ejaculation if the sphincter at bladder neck is injured
  - Impotence if the parasympathetic fibres are affected

- Urethral stricture
- **TUR syndrome (Dilutional hyponatremia or water intoxication).**
  - » Occurs due to excessive absorption of hypotonic irrigating solution
  - » More chance of occurrence when operating time exceeds 90 minutes and gland size is > 75 gm
  - » Clinical features are of hyponatremia and treatment is hypertonic saline for severe symptoms and diuresis and fluid restriction for mild cases.
- **Open prostatectomy**
  - Indications of prostatectomy are the same as for endoscopic procedures.
  - **Specific indications for open procedure:**
    - Prostate more than 75gms,
    - Large bladder diverticula,
    - Large vesical calculi not amenable to treatment by endoscopic route.
  - **Contraindications:**
    - Prostate cancer
    - Bladder cancer
    - Small fibrous gland.
  - **Preoperative evaluation:** As above.
  - **Anesthesia:** GA/SA/epidural
  - **Types:**
    - **Millin's** retro pubic prostatectomy
    - **Frayer's** suprapubic transvesical prostatectomy
    - **Young's** perineal prostatectomy.
  - **Complications:**
    - Haemorrhage
    - UTI
    - Epididymitis
    - Incontinence, erectile dysfunction
    - Urethral stricture
    - Wound infection
    - Incisional hernia

**Q29. Enumerate the causes of acute urinary retention. Discuss the management of postoperative urinary retention.**

**Write the etiology, diagnoses and management of a case of 60 year old male with acute urinary retention.**

**Ans.** Acute urinary retention causes:

<b>Males</b>	<ul style="list-style-type: none"> <li>• Bladder outlet obstruction: BPH, carcinoma prostate</li> <li>• Phimosis</li> <li>• Meatal stenosis</li> <li>• Postoperative</li> <li>• Urethral stricture</li> <li>• Acute prostatitis</li> </ul>
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<b>Females</b>	<ul style="list-style-type: none"> <li>• Retroverted gravid uterus</li> <li>• Multiple sclerosis</li> </ul>
<b>Males and females</b>	<ul style="list-style-type: none"> <li>• Faecal impaction</li> <li>• Post haemorrhoidectomy, pelvic surgery like APR</li> <li>• Blood clot in the bladder</li> <li>• Neurogenic bladder</li> <li>• Urethral calculus</li> <li>• Aging leading to smooth muscle dysfunction</li> <li>• Postoperative analgesia</li> <li>• Post spinal anesthesia</li> <li>• Drugs: Antihistaminic, anticholinergic, tricyclic anti depressant</li> </ul>

### **Management of postoperative retention:**

Important causes: Pain, sedation, pelvic surgery, BPH in males.

### **Important management issues:**

- Patient re-assurance
- Analgesia
- Ensure patient privacy to void
- Sound of running water by making patient stand near a running tap of water
- Make patient sit on edge of bed—males
- Warm bath
- Catheterization if none of the above fails. Usually the patient has transient retention that is relieved on its own as the effect of anesthesia and drugs wean off. If the retention is persistent, then keep patient catheterized and do cystoscopy, catheter trial for bladder sensation before attempting extubation.

### **A case of 60 year old male with acute urinary retention.**

#### **Etiology**

- Benign prostatic hypertrophy
- Bladder neck stenosis
- Bladder neck hypertrophy
- Prostate cancer
- Urethral strictures
- Neuropathic bladder

#### **Management:**

- The patient is catheterized to relieve obstruction. If the catheter cannot be passed, suprapubic cystostomy should be done to relieve obstruction
- The renal function and serum electrolytes are monitored to diagnose post obstructive diuresis
- A detailed history is taken and DRE is done to see prostatic enlargement
- Ultrasonography KUB is done
- In case of BPH, patient is started on alpha blockers and trial of catheter removal is given after 48 to 72 hrs of starting the drug
- In case of neuropathic bladder, a urodynamic evaluation, neurogenic evaluation is done and further management done as per the reports.

**Q30. Write a note on PSA.**

**Ans.** PSA is a product of the prostatic epithelium and is secreted in semen. It is a serine protease and it liquefies the seminal coagulum after ejaculation.

Normal value	< 4 ng/ml
Common range for both BPH and malignancy	4 – 10 ng/ml
75-80% chance of malignancy	10 – 35 ng/ml
Diagnostic of malignancy	> 35 ng/ml

**Limitation**

- It is organ specific and its level correlates with tumor burden but it is not cancer specific. Conditions that can raise PSA level are as follows:
  - BPH and cancer prostate
  - Ageing
  - Ejaculation
  - Prostatitis
  - DRE, Instrumentation or catheterization near prostate
  - Infarcts in prostate
- 20–25% patients even with prostate cancer can have PSA level < 4 ng/ml.

**Refinements in PSA to avoid diagnostic difficulties**

- **PSA density**
  - PSA produced/gram of prostatic tissue
  - Serum PSA/ Volume of prostate
  - Value < 0.15 is consistent with BPH
  - Value > 0.15 is more suggestive of malignancy.
- **PSA velocity**
  - Rate of change of PSA with time
  - 3 to 4 PSA measurements in a period of 2 years is necessary to calculate this value
  - Normal—0.75 ng/ml/year
  - Greater than normal suggests malignancy.
- **% of free PSA**
  - Free PSA 100/Total PSA = The value is lower in malignancy than in BPH
  - It is the most valuable marker to differentiate between malignancy and BPH when Serum PSA is in the problem zone of 4 to 10 ng/ml
  - Values less than 10 are more likely to be suggestive of malignancy.
- **Immunoreactive PSA**—Bound to alpha-1 antichymotrypsin is the major fraction where as the free fraction is the minor fraction.

**Best measure**

- Serum PSA + DRE + Transrectal Radiology (USG or MRI) are the best measures to be certain to make a confirm and early detection of prostatic carcinoma till the refinements are standardized.

**Uses**

- Cancer diagnoses
- Serial measurements of PSA levels are of value in assessing response to treatment.



## TESTIS AND SCROTUM

### Q31. Discuss the development of testis.

Ans.

- Testes develops in the lumbar region in retroperitoneum from genital ridge medial to mesonephros
- It is attached to the posterior abdominal wall by urogenital mesentery called mesorchium
- Caudal portion of the mesorchium is called genital ligament
- Also, gubernaculum extends from the caudal pole of testes and extend initially upto extra-abdominal portion between the developing internal and external obliques
- Later, it migrates towards scrotal swelling and finally attaches at the scrotal floor
- Intraabdominal migration of testes towards scrotum occurs due to differential growth rates of gubernaculum and abdominal cavity
- Increased intraabdominal pressure aids in the migration through the inguinal canal
- Regression of the extraabdominal portion of the gubernaculum leads to the final position of testes
- Hormones such as chorionic gonadotrophins, mullerian inhibiting substance and androgens also aid in the descent
- **Levels at different gestational ages are as follows:**

<b>3 months</b>	Iliac fossa
<b>Early 7<sup>th</sup> month</b>	Deep inguinal ring
<b>End of 7<sup>th</sup> month</b>	Traverse through the inguinal canal
<b>8 months</b>	Superficial inguinal ring
<b>9 months</b>	Scrotum at its final position

Associated with this but independent from it is the evagination of a fold of peritoneum that follows the course of gubernaculum and forms the processus vaginalis.

- **Factors hindering normal descent:**
  - Testicular agenesis or dysgenesis
  - Short testicular vessels/pampiniform plexus/Vas deferens
  - Hormone deficiency
  - Retroperitoneal adhesions.

### Q32. What is cryptorchidism? Discuss its management.

**What is undescended testis? Differentiate it from ectopic testis and write its management outline.**

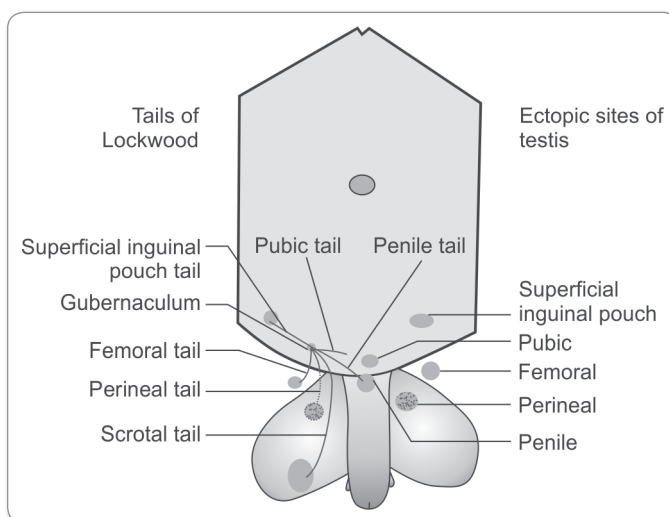
Ans.

- Cryptorchidism is basically absent testes in scrotum with unidentified position of testes
- Write the development of testes from that question
- **The cryptorchid testes can be undescended testes or ectopic testes**
- **Undescended testes**
  - It means the testes has stopped somewhere in its normal pathway of descent

- Undescended testes can be abdominal, iliac, retractile (Superficial inguinal—deep to the external oblique aponeurosis), emergent (Inguinal canal), simple undescended testes (Just above its usual position in the upper regions of scrotum)
- Incidence of undescended testis is 3% at birth and 1% by 1 year
- Of these, 80% are palpable and 80% normally descend by 3 to 4 months of age
- Epididymal anomalies are present in most of these cases and renal anomalies can be associated
- It is more common on the right side because of later descent of right testis compared to left.
- **Ectopic testes**
  - It means the testes has descended but does not reach its anatomical position and instead reaches some place that it does not usually belong to
  - Ectopic testes arises because of abnormal insertion of gubernaculums which can be superficial inguinal, pubic, perineal or femoral from above downwards according to the tails of lockwood.

### Differentiating points

Undescended testes	Ectopic testes
More common	Less common
Scrotum of affected side is less developed	Scrotum is normally developed
Testes is deep to the external oblique aponeurosis and therefore on tensing the muscle, it becomes deep and less mobile	Testes is superficial to the external oblique aponeurosis and therefore on tensing the muscle, it becomes more prominent and remains mobile
Occurs due to arrest of normal descent	Occurs due to abnormal gubernaculums insertion
Types are as described above	Types are as described above
In retractile undescended testes, the testes can be brought into scrotum by gentle manipulation	In ectopic testes, the testes cannot be brought into scrotum



**Fig. 4:** Ectopic testes outline

**Complications arising in cryptorchid testes**

- Atrophic testis
- Infertility
- Malignancy/torsion testis/trauma
- Epididymo-orchitis
- Herniation of testis.

**Investigations**

- Diagnostic laparoscopy is the investigation of choice
- MRI is the best noninvasive investigation
- Presence of blind ending testicular artery is the most definitive investigational finding of absent testis.

**Treatment**

- Timing of surgery = At 6 months age and maximum by 1 year age.

**Role of laparoscopy**

- Detect abdominal/high abdominal testis (> 2.5 cm from the deep ring)
- Detect absent/atrophic testis
- Laparoscopic Fowler Stephen procedure is not being done
- Can do bilateral surgery simultaneously.

**Procedures (FOL<sup>2</sup>KS<sup>2</sup>)**

- Fowler-Stephen 1 stage or 2 stage procedure
- Ombredanne technique
- Ladd and Gross technique
- Laparoscopy
- Keetley-Torrek technique
- Subcutaneous dartos pouch placement
- Silber microvascular anastomosis of testicular vessels to inferior epigastric vessels
- Medialization of cord structures by division of the inferior epigastric and deep ring structures is last resort in cases where the length is not adequate for putting the testis in normal position or in subcutaneous dartos pouch
- In cases with bilateral undescended testis, human chorionic gonadotrophin therapy is given for a month to see if testosterone, LH and FSH increases or not. If it does not increase, it means anorchia whereas if it increases, laparoscopy is done to find the testis and do appropriate surgery to take care of the undescended testis.

**Q33. What is testicular torsion? Discuss its differential diagnoses.****Discuss the management of testicular torsion.**

**Ans.**

**Pathophysiology**

- **Predisposing factors:** Inversion of testis, bell clapper deformity due to high investment of tunica vaginalis
  - **Extravaginal testicular torsion** occurs when the the spermatic cord and tunica vaginalis are fused and rotate as a unit. It usually occurs in the perinatal period

- **Intravaginal torsion, or torsion of the cord** within the visceral and parietal layers of tunica vaginalis. The space between the parietal and visceral layers of the tunica vaginalis extends proximally upto the cord for a variable distance and creates an abnormally mobile testis that hangs freely within the created space (“**bell-clapper deformity**”)
- Sudden contraction of the cremasteric muscle, which inserts onto the cord in a spiral configuration is the initiating event.

**Clinical features:**

- Acute onset scrotal/testicular pain that may radiate to the lower abdomen
- Constitutional symptoms may be present
- On examination, the ipsilateral testis/scrotum is very tender, tense and oedematous
- The testis is high hiding and may have a transverse lie
- On derotating the testis, there is sudden relief of symptoms
- The ipsilateral cremastic reflex is absent.

**Diagnosis:**

- Clinical examination is enough to diagnose
- Ultrasonography and Doppler of the scrotum show absent flow in the ipsilateral testis.

*Differential diagnosis of testicular torsion:*

- Torsion of the appendix of testis (Peduncular hydatid of Morgagni). Ultrasound shows blue dot sign. Conservative management is enough if the diagnoses is confirmed
- Torsion of the appendix of epididymis
- Torsion of the spermatic cord
- Epididymo-orchitis (Prehn sign and cremasteric reflex are negative)
- Strangulated inguinal hernia
- Idiopathic scrotal edema (Allergic phenomenon associated with eosinophilia. Subsides on its own)
- Nonurogenital pathology (e.g., adductor tendinitis)
- Hematocele
- Mumps orchitis (No cord thickening, usually bilateral).

**Management:**

- Surgery is the treatment of choice and should be done within 4 to 6 hours of diagnoses to prevent testicular gangrene and atrophy
- Bilateral surgery is to be done
- A median raphe scrotal incision may be used to explore both sides
- The affected side should be examined first and the cord detorsed to reestablish blood flow to the testis
- Testes with marginal viability should be placed in warm sponges and re-examined after several minutes and preserved if normal or resected if necrotic. If the testis is to be preserved, it should be placed in the dartos pouch with suture fixation
- **The contralateral testis must be fixed to prevent subsequent torsion.**

**Q34. What is epididymo-orchitis? Differentiate it from torsion testis and Discuss its management.**

**Ans.**

- Epididymo orchitis refers to the inflammation of testis and epididymis from ascending infection of the urinary tract.

**Etiopathogenesis**

- Can occur in any age group. More commonly due to a sexually transmitted infection in young males and due to ascending urinary infection in children or adults
- Organisms: *Chlamydia*, *N gonorrhoea* in sexually active males and *E. Coli* in children and elderly male.

**Clinical features:**

- Urinary frequency
- Dysuria
- Burning micturition
- Fever
- On examination: Epididymis and testis are tender, swollen, enlarged, cord thickened, and presence of reactive hydrocele
- Elevation of testis relieves pain (Prehn test positive).

**Investigations**

- Urine analysis: Pyuria, bacteria, positive leucocyte esterase
- Urine culture
- Ultrasound scrotum.

**Treatment:**

- Antibiotics
- Scrotal support
- Analgesics also act as anti-inflammatory agents and provide symptomatic relief.

**Differences between epididymo-orchitis and testicular torsion are as follows:**

Testicular torsion	Epididymo-orchitis
Prepubertal age group	Adults 20-30yrs
Sudden agonizing pain, high riding testis, transverse lie of testis	Swollen, red testis, tender scrotum, thickened cord, reactive hydrocele, normal lie of testis
Shorter duration of symptoms	Longer duration of symptoms
Cremastic reflex absent Prehn sign negative	Cremasteric reflex Present Phren sign positive
Urine analysis: Normal	Urine analysis: Pyuria, bacteriuria
Doppler: Decreased blood flow	Doppler: Increased blood flow
Treatment: Surgical emergency—immediate scrotal exploration	Conservative management

**Q35. Q. write a note on varicocele.**

**Ans.** Varicocele refers to dilatation and tortuosity of pampiniform plexus of veins.

**Types**

- **Primary**—Idiopathic. It has no underlying etiology.
- **Secondary**—Has some underlying etiology such as renal cell carcinoma, pelvic tumors etc.

**Clinical features**

- Dragging sensation, pain, infertility
- The swelling feels like a bag of worms on palpation
- Cough impulse present
- Failure of varicocele to decompress after patient lies supine is suspicious for secondary varicocele like associated RCC with invasion into renal vein.

**Grading of varicoceles**

- **Subclinical:** Visualized only on Doppler
- **Grade I:** Palpable only with the valsalva maneuver
- **Grade II:** Palpably detected without the valsalva maneuver but not visible at rest
- **Grade III:** Visible through the scrotal skin when the patient is at rest.

**Investigation:**

- Physical examination remains the gold standard for diagnosis
- Semen analysis in cases of infertility
- Doppler ultrasound is confirmatory. There is reversal of flow in pampiniform plexus with valsalva along with dilatation of veins to more than 3.5 mm in diameter.

**Indications for surgery:**

- An infertile adult man with a varicocele when couple has known infertility and the female partner has normal fertility or a potentially treatable cause of infertility with the varicocele palpable on physical examination or ultrasound and the male partner has an abnormal semen analysis
- Reduction in ipsilateral testicular volume by more than 20% or 2 ml as compared to contralateral normal size
- Army or navy recruitment.

**Surgical options**

- **Scrotal approach:** Obsolete
- **Retroperitoneal approach: Palomo approach:**
  - A transverse abdominal incision is made at the level of the internal inguinal ring, approximately two fingerbreadths medial to the anterior superior iliac spine and opened in layers
  - The peritoneum is reflected medially, and the muscles are retracted cephalad to expose the internal spermatic vessels

- The internal spermatic vein is then ligated and divided, proximal to its insertion into the left renal vein
- **Complications:** Recurrence, hydrocele
- **Inguinal approach (Modified Ivanissevich)**
- **Subinguinal approach:** Similar to inguinal approach but difficult as there are far more number of branches below the deep ring and hence some of these may be missed leading to recurrence
- **Percutaneous embolization:** Least invasive methods wherein these dilated veins are embolized using coils by a percutaneous technique.

**Q36. Enumerate the types of hydrocele and discuss the differences between primary and secondary hydrocele.**

**Ans.** Accumulation of fluid between the two layers of tunica vaginalis is called as hydrocele.

**Types of hydrocele**

<b>Vaginal hydrocele</b>	Limited to the tunica covering the testis
<b>Funicular hydrocele</b>	Patent processus upto the top of testis
<b>Infantile</b>	The tunica and processus vaginalis are distended upto the inguinal ring without any connection with the peritoneal cavity
<b>Congenital hydrocele</b>	Patent processus vaginalis is present which allows the peritoneal cavity to communicate freely with the tunica covering the testis
<b>Hydrocele en bisac/ bilocular hydrocele</b>	There are two intercommunicating sacs one above and one below the neck of scrotum. There is no intra peritoneal communication with the upper sac
<b>Encysted hydrocele of the cord</b>	Central portion of the processus vaginalis is patent with obliterated proximal and distal portion
<b>Hydrocele of canal of nuck</b>	Related to the round ligament in females and is the counter part of encysted hydrocele of the cord

**Differences between primary and secondary hydrocele are as follows**

<b>Primary hydrocele</b>	<b>Secondary hydrocele</b>
Idiopathic	Secondary to a predisposing cause like epididymo-orchitis, testicular cancer
Larger in size	Smaller
Tense	Lax
Transillumination positive	Usually Negative
Testis usually not palpable	May be palpable
Doesn't resolve spontaneously	May resolve with treatment of underlying condition like epididymo-orchitis

**Q37. Write a note on Fournier gangrene.**

**Ans.**

- Fournier gangrene is a synergistic gangrene (Other synergistic gangrene is Meleney's gangrene of abdominal wall)

- This means that it is caused by a combination of both aerobic and anaerobic infection
- It is a form of necrotizing fasciitis of scrotum that involves the entire scrotum rapidly but spares the testis and cord structures
- It is more common in patients with immunosuppression such as AIDS, IV drug use, chemotherapy, steroid therapy, local trauma or surgery, diabetes mellitus, alcoholism, malnutrition and local infections in that area
- Fulminant infection results in endarteritis and rapid spread along fascial planes lead to gangrene of the scrotal skin and fascia
- Spread into blood stream and lymphatics lead to the features of septicaemia and septic shock
- It is a highly lethal condition with nearly 50–60% mortality.

### Clinical presentation

- Scrotal gangrene
- May start as cellulitis and then develops into overt gangrene
- Signs of necrotizing fasciitis on scrotal skin such as dishwater pus, pain out of proportion of apparent clinical involvement of skin, crepitus
- Fever, features of septicemia.

### Investigations

- Blood culture
- Urine culture
- Local discharge culture
- Other routine investigations.

### Treatment

- Urgent radical debridement is life saving
- Start on penicillin and other antibiotics as deemed necessary and once culture is received, based on culture—sensitivity report
- IV fluid resuscitation and electrolyte and acid base management should also proceed simultaneously
- Multiple debridements might be required to control the infection
- Final repair is done usually by implantation of the testis in thigh pockets and plastic surgical repair of the scrotum.

### Q38. Write the differential diagnoses of a scrotal swelling.

**Ans.** The differential diagnoses of a scrotal swelling is as follows:

Hydrocele	
Haematocele	<ul style="list-style-type: none"> <li>• Occur following tapping percutaneously of a hydrocele, or after external trauma</li> <li>• Patient presents with a scrotal swelling</li> <li>• Acute ones are painful</li> <li>• Old hematocele may present similar to testicular tumor</li> <li>• Absence of gradual testicular enlargement, presence of testicular sensation and absence of metastasis favours a clinical diagnosis of hematocele</li> </ul>

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<b>Pyocele</b>	<ul style="list-style-type: none"> <li>Occurs secondary to infection in a hydrocele</li> <li>The overlying skin is healthy</li> <li>The patient has fever and other constitutional symptoms of infection</li> </ul>
<b>Gumma testis</b>	<ul style="list-style-type: none"> <li>Caused by syphilis</li> <li>It may present with bilateral orchitis (congenital syphilis)</li> <li>Interstitial fibrosis (symptomless, bilateral, no or mild testicular enlargement, testicular sensation is lost)</li> <li>Gumma (unilateral, gradual testicular enlargement, painless, testis becomes hard with loss of testicular sensations, associated with hydrocele)</li> </ul>
<b>Filarial epididymo orchitis</b>	<ul style="list-style-type: none"> <li>Testis is enlarged, firm, mildly tender and partial loss of testicular sensation occurs</li> <li>Epididymitis and secondary hydrocele can occur</li> <li>The groove between the testis and epididymis is obliterated by inflammatory infiltrates</li> </ul>
<b>Tubercular orchitis</b>	<ul style="list-style-type: none"> <li>Testis is enlarged, slight tender, accompanied by epididymal enlargement and loss of scrotal rugosities</li> <li>Spermatic cord/ vas deferens may have a nodular feel due to submucousal nodules</li> <li>Cold abscess may form on posterior aspect of epididymis</li> </ul>
<b>Chylocele</b>	<ul style="list-style-type: none"> <li>Suspected if a patient presents with history of fever</li> <li>Patient is usually a resident of an endemic area</li> <li>Fluid may demonstrate microfilariae</li> </ul>
<b>Lymph varix</b>	<ul style="list-style-type: none"> <li>Associated with filariasis</li> <li>It is dilatation and tortuosity of lymphatics of spermatic cord</li> </ul>
<b>Spermatocele</b>	<ul style="list-style-type: none"> <li>Unilocular cystic swelling located in head of epididymis</li> <li>Fluid contains spermatozoa and resembles barley water</li> </ul>
<b>Testicular tumor</b>	<ul style="list-style-type: none"> <li>Painless enlargement of testis</li> <li>Testis is hard on palpation, with loss of testicular sensation</li> <li>Symptoms of metastases may be present</li> <li>It may present with a secondary hydrocele</li> </ul>
<b>Other uncommon differentials</b>	<ul style="list-style-type: none"> <li>Cysts of hydatid of morgagni</li> <li>Varicocele</li> </ul>

### Q39. Write a note on phimoses and paraphimoses.

Ans.

#### Phimosis

It is the inability to retract the prepuce.

#### Causes

- Congenital
- It is physiological upto five years of age
- Acquired
  - Recurrent attacks of balanoposthitis or balanitis
  - Malignancy
  - Balanitis xerotica obliterans.

*Clinical features*

- It can cause ballooning up of prepuce while passing urine
- It can also lead to recurrent balanitis and balanoposthitis.

*Treatment*

- Topical corticosteroids
- Circumcision
- Dorsal slit.

**Paraphimosis**

- It is an acquired condition
- The prepuce is retracted back and due to oedema, it cannot be repositioned back if not repositioned initially
- It most commonly results from unknowingly failing to reposit back the prepuce after retracting it for a procedure like catheterization
- Oedema of prepuce leads to swelling, venous congestion further oedema, arterial compromise, formation of constriction ring around the base of glans.

*Treatment*

- Application of hygroscopic agent like glycerine to reduce oedema, manual reposition by applying pressure on glans and counter pressure on prepuce
- Circumcision in cases where above methods fail.

**Q40. Write a note on Vasectomy.****Ans.**

- It is a method of male sterilization/contraception
- It should be considered as a permanent method of sterilization
- It is performed as an outpatient procedure under local anesthesia and bilateral at the same time.

**Counseling before procedure**

- Patients should be told that it is a permanent procedure
- Spontaneous recanalization is a known entity
- Incidence of chronic testicular pain is 5%
- Abstinence or other method of contraception is to be used for 12 to 16 weeks till semen examination is done after 12 to 16 weeks of the procedure or till 20 ejaculations
- Reversal has potency rates of up to 80% but fertility rates are very low because of secondary testicular failure after vasectomy and the development of anti-sperm antibodies.

**Types**

- Traditional vasectomy: 1 cm midline scrotal or bilateral scrotal incisions are used
- No Scalpel Vasectomy: First done in China in 1974.

**Important steps**

- Preoperative shaving
- Antibiotic prophylaxis is not recommended

- No scalpel procedure needs sharp pointed artery forceps to make a midline scrotal puncture and Allis or Ring forceps is used to fix and deliver vas from the wound
- Once this is done, remove all the coverings of the vas and reapply ring forceps
- Divide vas, resect a 1 cm segment 3 cm distal to the end of epididymis and tie both ends with vicryl/ clips with burying of the cut ends or turning them back on themselves or cauterise the lumens or separate the ends in different tissue planes to prevent spontaneous recanalization
- Silk ties are avoided as it can cause sinus formation.

**Postoperative care**

- Limited heavy or strenuous activity for 1 week
- NSAIDs are required if not contraindicated
- Cold compresses for 1–2 days.

**Benefits**

- Decreased hematoma, pain, infection at wound site
- Shorter operative time
- No incision therefore no incision related complications.

**Complications**

- Hematoma
- Infection (Fournier gangrene)
- Scrotal sinus/fistula
- **Chronic scrotal pain (Postvasectomy pain syndrome)**
  - Scrotal elevation and scrotal support, heat or ice application and oral NSAIDs is the first line treatment
  - Second line treatment: Spermatic cord blocks and excision of sperm granulomas
  - Third line treatment: Vasectomy reversal
  - Last resort to severe intractable pain: Orchiectomy (Inguinal orchiectomy has better response rates than scrotal orchiectomy).

**Q41. Enumerate the indications of circumcision and write its steps.**

**Ans.**

**Indications**

- Phimosis
- Paraphimosis
- For taking biopsy from prepuce or glans
- Prior to radiotherapy for penile cancer
- Religious
- In neonates on desire of parents.

**Contraindications**

- Neonates with hypospadias
- Patients with chordee without hypospadias
- Dorsal hood deformity
- Webbed penis, or small penis.

**Steps**

- Consent checked. Patient is laid supine. Part cleaned and draped
- General anesthesia is preferred in infants
- Local anesthesia is given in other patients-Penile block and local infiltration along the corona
- Three clamps are applied at 12, 3 and 9 o'clock position of tip of prepuce. The prepuce is kept at stretch
- Incision is given vertically at 12 o'clock position till the corona glandis. The prepuce is excised by giving a circumferential incision at corona. The skin and mucosa at corona are sutured together with an absorbable suture like catgut
- Hemostasis secured and gentle dressing applied.

**Complications**

- Bleeding
- Meatal stenosis
- Removal of too much or too little skin
- Wound infection
- Penile adhesions
- Injury to glans, urethra, penile shaft.

## PENIS AND URETHRA

**Q42. Discuss the differential diagnoses of a patient with a penile ulcer.**

**Ans. The cause of penile ulcer can be any of the following:**

- Syphilitic chancre (*Treponema pallidum*)
- Chancroid (*Hemophilus ducreyi*)
- Genital herpes (Herpes simplex virus 2 > 1) – most common cause
- Lymphogranuloma inguinale (*Chlamydia Trachomatis* A L1-3)
- Granuloma inguinale (*Calymmatobacter granulomatis* or *klebsiella granulomatis*)
- Penile carcinoma (Ulceroproliferative squamous cell carcinoma)
- Traumatic ulcer.

**Differentiating features**

Ulcer <b>without</b> inguinal lymphadenopathy	Granuloma Inguinale	<ul style="list-style-type: none"> <li>• Painless ulcer with beefy red base which bleeds to touch</li> <li>• Can have inguinal pseudobubo and lower limb pseudoelephantiasis</li> <li>• Keloid and malignant change are common complications</li> </ul>
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<b>Painless ulcer with multiple bilateral painless</b> inguinal lymph nodes (- -)*	Syphilis	<ul style="list-style-type: none"> <li>• Ulcer has indurated base</li> <li>• Punched out margins and wash leather slough in base</li> </ul>
<b>Painful ulcer with single painful</b> inguinal lymph node (+ +)*	Chancroid	<ul style="list-style-type: none"> <li>• Ulcer is single or multiple with undermined edge</li> <li>• The involved lymph node is called bubo</li> </ul>
<b>Painless ulcer with multiple painful</b> inguinal lymph nodes (- +)*	Lymphogranuloma venereum	<ul style="list-style-type: none"> <li>• Usually single ulcer</li> <li>• <b>Groove sign of Greenblatt</b> is present due to enlarged lymphnodes producing a swelling on both above and below the inguinal ligament with the ligament appearing as a groove in the swelling</li> <li>• No systemic symptoms</li> </ul>
<b>Painful Ulcer with multiple painful</b> inguinal lymph node [+ +]*	Herpes	<ul style="list-style-type: none"> <li>• Multiple shallow ulcers</li> <li>• Systemic symptoms are usually present</li> </ul>

[\* = (pain in ulcer pain in lymph node)]

Disease	Diagnostic tests	Treatment
Herpes	PCR Cell culture Clinical examination findings	Acyclovir 400 mg TDS oral for 7–10 days
Syphilis	Dark ground microscopy Serological tests	Benzathine penicillin G 2.4 million units IM single dose Repeat the above dose weekly for 3 more doses if tertiary syphilis
LGV	Serological tests clinical examination findings	Doxycycline 100 mg BD for 3 weeks Erythromycin 500 mg QID for 3 weeks
Granuloma inguinale	Ulcer edge biopsy and microscopy shows gram negative rods in large multinucleated cells	Doxycycline 100 mg BD for 3 weeks Erythromycin 500 mg QID for 3 weeks
Chancroid	Ulcer edge biopsy and microscopy Serological tests	Single dose Azithromycin 1 gm oral Single dose ceftriaxone 250 mg IM

**Q43. Discuss the clinical features and management of urethral injury in brief.****Ans.**

- Mostly in males, after straddle injuries or pelvic fracture
- Urethra divided into: **Posterior urethra** (prostatic and membranous urethra) and **anterior urethra** (bulbous urethra and penile urethra).

**Posterior urethral injury:**

- Membranous urethra or more specifically the bulbomembranous junction is the mostly injured site after pelvic fracture
- Urinary extravasation may occur into the retroperitoneum and pelvis.
- **Clinical features:**
  - Urinary retention
  - Blood at external urethral meatus
  - The prostate is high riding on digital rectal examination
  - Extraperitoneal bladder rupture is associated in 20–25% of the cases.
- **Immediate management**
  - **Retrograde urethrography** is the first step and is done to demonstrate urethral injury and its level
  - **IVP** shows butterfly perineal hematoma and “Pie in the sky appearance” of prostate.
  - In case of a palpable urinary bladder, a **suprapubic catheter** is inserted
  - Otherwise a **single gentle attempt of per urethral catheterization** may be attempted.
- **Definitive repair**
  - Partial tears or lacerations are healed by above methods (Catheter or SPC)
  - Complete tears and disruptions require endoscopic or surgical repair
  - Immediate definitive repair is abandoned in man due to increased incidence of postoperative stricture. It is still done in females
  - Delayed repair at 3–4 months after injury is the preferred approach
  - **Open posterior urethroplasty** through a perineal approach or **endoscopic internal urethrotomy** and repair under direct vision for defects <1cm are the two management options.

**Anterior urethral injury**

- Occurs due to direct blow to the perineum
- Other causes are straddle injury, self instrumentation or iatrogenic injury
- Mostly injury occurs in bulbar urethra
- Superficial urinary extravasation may occur into the scrotum, along the penile shaft and upwards into the abdominal wall
- **Clinical features:**
  - Urinary retention
  - Blood at external urethral meatus
  - Perineal hematoma
  - Normal position of prostate on digital rectal examination.
- **Management:**

Retrograde urethrography is done to establish extent of injury (contusion/laceration/disruption and urinary extravasation)

Suprapubic cystostomy is done to drain urine as an immediate measure

**Definitive repair** is done at a later stage after 3–4 months of injury with open anastomotic urethroplasty.

Anterior urethral injury	Posterior urethral injury
More common	Less common
Direct blow to perineum	Pelvic fracture
Signs and symptoms: Retention of urine, blood at meatus, perineal hematoma, with a normal position of prostate on DRE	Retention of urine, blood at meatus, pelvic hematoma and a high riding prostate on DRE
Urinary extravasation is superficial	Urinary extravasation is deep

**Q44. Enumerate the causes of urethral stricture and discuss its clinical features and management.**

**What are the complications of urethral stricture? Discuss its management.**

**Ans.**

- It is a disease of the anterior urethra
- It results from a scarring process involving the corpus spongiosum (spongiofibrosis), leading to narrowing of urethral lumen.

**Etiology**

- Congenital
- Trauma
- Inflammatory (gonorrhea, balanitis xerotica obliterans and lichen sclerosis)
- Ischemic
- Malignant
- Instrumental (post catheterization or endoscopy)
- Post operative (open prostatectomy, penectomy).

**Pathophysiology**

- Ischemia and fibrosis leads to scarring of the corpora spongiosum which contracts and leads to reduced calibre of lumen of urethra leading to resistance of urinary outflow and back pressure effects on the upper urinary tract.

**Clinical features**

- Obstructive voiding symptoms
- Urinary tract infections such as prostatitis and epididymitis
- Acute or chronic urinary retention with hydronephrosis and renal failure
- Urethral fistula or periurethral abscess.

**Investigations**

- **The length and location of the stricture**—urethroscopy, and ultrasonography
- **The depth and density of the scar**—physical examination, the appearance of the urethra in contrast-enhanced studies, and the amount of elasticity noted on urethroscopy
- Radiography includes **MCU/RGU** to delineate the proximal and distal aspects of the scar.

**Management**

- **Dilatation**
  - The goal of this treatment is to stretch the scar without producing more scarring and therefore no bleeding should occur during the procedure or after it.

- **Internal urethrotomy**
  - The procedure involves a transurethral approach and is basically an incision through the scar to healthy tissue to allow the scar to expand (release of scar contracture) and the lumen to heal enlarged
  - Complications include recurrence of stricture, rupture of urethra and bleeding.
- **Excision and re-anastomosis**
  - **For strictures less than 2 cm in length**—complete excision of the area of fibrosis, with a primary tension free spatulated reanastomosis is the procedure of choice.
  - **For strictures more than 2 cm in length**—tissue transfer techniques using skin graft, the bladder epithelial graft, the buccal mucosal graft, and the rectal mucosal graft are used to cover the defects created by complete excision.

**Q45. Write a note on lasers in urology.**

**Ans.**

- Laser stands for light amplification by the stimulated emission of radiation
- Laser is a thermal ablative technique. (Refer to the classification of ablative techniques in the section on surgical technology).

**Mechanism**

- Stimulated emission of radiation when a photon is stimulated by high intensity light
- A flashlamp gives out high-intensity light, which then bombards a resonator cavity with photons
- The electrons in the resonator cavity, which are excited by the bombardment of photons, are caused to jump to higher or “excited state” orbitals which leads to a very rapid decay of the electrons, which emit a photon. This process is known as spontaneous emission of radiation
- This emitted photon has the energy required to interact with other excited state atoms which propagate the reaction of further electron orbital decay and photon emission are induced
- This is known as stimulated emission of radiation
- These photons leave the resonator cavity as a coherent laser beam
- The LASER beam thus differs from the normal light because
  - It is monochromatic
  - It is collimated
  - It is coherent

**Types of laser** in common use:

Ho: YAG (2100 nm), Nd: YAG (1064 nm), KTP (532 nm), Diode (820 nm).

**Uses of laser:**

- Treatment of BPH
  - First system—Transurethral USG guided laser induced prostatectomy (TULIP)
  - The technique now used is Ho: YAG dual wavelength pulse select laser
- Minimally invasive Nd:YAG ablation of tumors upto 2.5 cm size in Ta, Tis urinary bladder tumors
- Laser interstitial thermal therapy (LITT) is under evaluation for advanced renal tumors using Nd:YAG or Diode



- HO:YAG laser intracorporeal lithotripsy for ureteric calculi breaks all stones irrespective of content and is the safest and most effective means
- KTP/Nd:YAG/Argon laser for treatment of ureteric strictures
- Fulgration of ureteral or renal pelvic or penile tumor
- Fulgration of posterior urethral valves
- Laser endoscopic pyelotomy in PUJ obstruction mainly in patients with small intrarenal pelvis and length of abnormality  $< \text{or} = 2 \text{ cm}$ .

**Q46. Write a note on posterior urethral valve.**

**Ans.**

- These are mucosal folds projecting from the prostatic urethra to the external urethral sphincter
- Called as valves as they allow only unidirectional flow across them, hence obstructs voiding but allow passage of instrument from urethra to bladder
- Occur 1 in 8000—1 in 25000 live births.

**Types (Young's classification):**

<b>Type 1 (m.c.)</b>	Urothelial fold at or just distal to verumontenum. It is actually a ridge at the floor of the urethra
<b>Type 2</b>	Arising from verumontanum and runs along the posterior urethral wall. It is not obstructive
<b>Type 3</b>	Just distal to verumontanum, it is a membrane lying transversely across urethra with a small perforation in the center

**Pathophysiology**

- Proximal urethra, prostate, bladder neck, bladder, ureters, and kidneys are all affected and suffer from various forms of damage
- Various associations and disease patterns with posterior urethral valve are as follows:

<b>Urinary bladder</b>	Urinary tract infection Palpable distended bladder Vesicoureteric reflux in 50% cases
<b>Ureter</b>	<ul style="list-style-type: none"> <li>• Hydronephroses</li> </ul>
<b>Kidney</b>	<ul style="list-style-type: none"> <li>• Renal failure due to glomerular injury</li> <li>• Nephrogenic diabetes insipidus</li> <li>• Obstructive uropathy</li> <li>• Renal parenchymal dysplasia is the most important prognostic indicator</li> </ul>
<b>Lungs</b>	<ul style="list-style-type: none"> <li>• Pulmonary hypoplasia—This is the most common reason for death in these newborns</li> </ul>
<b>Abdomen</b>	<ul style="list-style-type: none"> <li>• Ascites</li> </ul>
<b>Antenatal problems</b>	<ul style="list-style-type: none"> <li>• Oligohydramnios</li> <li>• Bilateral hydronephrosis</li> </ul>

**Clinical presentation**

- Mostly diagnosed on prenatal ultrasound with pulmonary hypoplasia, renal insufficiency, ascites
- Postnatally can present with any of the presentations as mentioned above.

**Diagnosis**

- **Ultrasonography:** Detects bilateral hydronephrosis, increased echogenicity of kidneys, bilateral hydronephrosis and distended urinary bladders
- **Voiding cystourethrography (VCUG):** Investigation of choice in diagnosis of posterior urethral valves because it defines the anatomy and gross function of the bladder, bladder neck, and urethra
- Serum creatinine, blood urea nitrogen and serum electrolytes.

**Management**

- Urinary catheter drainage is the first step
- Stabilization of medical condition and normalization of creatinine levels in serum
- Endoscopic laser fulguration of the valve is the procedure of choice after stabilization
- If the child cannot be stabilized and creatinine cannot be normalized, a cutaneous vesicostomy or upper urinary tract diversion is done.

**Q47. Write a note on proteinuria.**

**Ans.** Abnormal amounts of protein in the urine.

**Causes/ Types**

<b>Glomerular proteinuria (m.c.)</b>	<ul style="list-style-type: none"> <li>• Primary glomerular diseases (IgA nephropathy, membranous or membranoproliferative glomerulonephritis etc.)</li> <li>• Systemic glomerulopathy (diabetes, SLE, amyloidoses, nephrosclerosis)</li> <li>• Occurs due to increased glomerular capillary permeability to protein</li> <li>• Almost certain when 24 hour urinary protein is very high (&gt; 3 gm)</li> </ul>
<b>Tubular proteinuria</b>	<ul style="list-style-type: none"> <li>• Tubular dysfunction disorders that result in failure to reabsorb the normally filtered low molecular weight protein</li> <li>• 24 hour urinary protein is usually less than 2 gm</li> </ul>
<b>Overflow proteinuria</b>	<ul style="list-style-type: none"> <li>• Multiple myeloma, myoglobinuria, Hemoglobinuria</li> <li>• Occurs because of increased plasma concentration of normally filtered low molecular weight protein that exhausts the tubular absorptive capacity</li> </ul>
<b>Other varieties</b>	<ul style="list-style-type: none"> <li>• <b>Benign orthostatic proteinurea</b></li> <li>• <b>Proteinurea with hematuria</b> is mostly glomerular cause</li> </ul>

**Range of proteinurea and extent of disease:**

<b>Normal proteinurea</b>	<ul style="list-style-type: none"> <li>• <b>&lt; 30 mg/24 hr urine</b></li> <li>• Contain 60-80% Tamm Horsfall protein and around 20% albumin</li> </ul>
<b>Microalbuminuria</b>	<ul style="list-style-type: none"> <li>• <b>30 – 300 mg/ 24 hr urine</b></li> <li>• Seen in diabetes nephropathy, hypertensive renal disease, patients on oral contraceptive pills or hormone replacement therapy</li> </ul>
<b>Macroalbuminuria</b>	<ul style="list-style-type: none"> <li>• 300 mg – 3 gm/ 24 hr urine</li> </ul>
<b>Nephrotic range proteinuria (Glomerular)</b>	<ul style="list-style-type: none"> <li>• &gt; 3 gm/24 hr urine</li> </ul>

**Detection techniques:**

<b>Qualitative detection</b>	<ul style="list-style-type: none"> <li>• Dipstick impregnated with tetrabromophenol blue dye</li> <li>• Heat and acetic acid test</li> <li>• Sulphosalicylic acid test</li> <li>• Protein electrophoresis and immunoelectrophoresis: Glomerular proteinuria is mainly albumin whereas tubular proteinuria is mainly immunoglobulins which can be differentiated by this test</li> </ul>
<b>Quantitative detection</b>	<ul style="list-style-type: none"> <li>• Esbach's albuminometer test</li> <li>• Biuret test</li> <li>• Protein-creatinine ratio</li> </ul>

**Management classification**

<b>Transient proteinuria</b>	<ul style="list-style-type: none"> <li>• Result from fever, exhaustive exercises, congestive heart failure, emotional duress and usually resolves spontaneously</li> <li>• No further evaluation is necessary unless it persists</li> </ul>
<b>Intermittent proteinuria</b>	<ul style="list-style-type: none"> <li>• Orthostatic proteinuria falls into this category and occurs secondary to pressure on renal vein on standing</li> <li>• In these patients, if the renal function test is normal, it does not need further evaluation</li> </ul>
<b>Persistent proteinuria</b>	<ul style="list-style-type: none"> <li>• Here 24 hour urine protein and protein electrophoresis should be done to differentiate glomerular from the tubular cause</li> <li>• If patient has more than 3 gm/24 hr, it is glomerular. Then if patient has hematuria, evaluate as for hematuria (discussed in question on hematuria)</li> <li>• Patients with proteinuria without hematuria should be evaluated for systemic causes of proteinuria</li> <li>• For protein 1-3 gm/24 hr, do immunoelectrophoresis for diagnoses of specific cause of tubular proteinuria or overflow proteinuria</li> <li>• Specific protein identification such as Bence Jones protein in multiple myeloma and hemoglobin or myoglobin in hemoglobinuria or myoglobinuria</li> </ul>

**Q48. Write a note on causes, investigations and management of male infertility.**

**Ans.** Traditional definition: Absence of conception after 12 months of unprotected intercourse.

WHO definition: Absence of conception after 24 months of unprotected intercourse.

**Incidence:** 15–20%.

**Incidence of male factor infertility** is 20% of all cases.

**Etiology**

<b>Varicocele</b>	<ul style="list-style-type: none"> <li>• Most common cause (4% cases)</li> </ul>
<b>Idiopathic</b>	<ul style="list-style-type: none"> <li>• 2nd most common cause</li> </ul>
<b>Obstructive cause</b>	<ul style="list-style-type: none"> <li>• Elective vasectomy</li> <li>• Iatrogenic injury during hernia repair, orchidopexy, Bladder neck/prostate surgery, retroperitoneal/pelvic surgery</li> <li>• Cystic fibrosis with vasal agenesis</li> <li>• Tubercular or syphilitic epididymitis</li> <li>• Mumps epididymo-orchitis</li> </ul>

*Contd...*

Contd...

<b>Testicular cause</b>	<ul style="list-style-type: none"> <li>• Congenital: Kallman syndrome, Primary hypogonadism</li> <li>• Testicular trauma/Torsion/Tumor</li> <li>• Radiation toxicity</li> </ul>
<b>Pretesticular cause</b>	<ul style="list-style-type: none"> <li>• Diabetes</li> <li>• Hodgkin's disease</li> <li>• Spinal cord injury</li> <li>• Multiple sclerosis</li> <li>• Hyperprolactinemia</li> <li>• Thyroid abnormalities (Both hypo and hyper)</li> </ul>
<b>Immunological</b>	<ul style="list-style-type: none"> <li>• Presence of antisperm antibodies</li> <li>• Immotile cilia syndrome</li> </ul>
<b>Drugs</b>	<ul style="list-style-type: none"> <li>• MAO inhibitors</li> <li>• Spironolactone, ketoconazole</li> <li>• 5 alpha reductase inhibitors</li> <li>• Exogenous steroid supplementation</li> </ul>

**Assessment**

- Both the husband and wife should be assessed simultaneously
- History and physical examination in relation to the above causes should be done
- **Semen analysis**
  - To be done within 2–3 hours of sample collection
  - To be examined for 2–3 samples
  - Normal values:

<b>Volume</b>	2-6 ml
<b>Colour</b>	White/Opalescent
<b>pH</b>	7.35 – 7.5
<b>Specific gravity</b>	1.028
<b>Sperm count</b>	> Or = 20 million/ ml. Less than this is called oligozoospermia and no sperms is called azoospermia
<b>Motility</b>	> Or = 50% or > Or = 25% progressive motility.
<b>Viability</b>	> Or = 50%
<b>Morphology</b>	> 30% normal morphology
<b>Pus cells</b>	< 1 × 10 <sup>6</sup> / ml

- **Endocrine testing**
  - LH, FSH, Thyroid testing, Prolactin, Testosterone
  - Azoospermia with testicular failure = FSH raised
  - Azoospermia with obstructive cause = FSH normal
- **Testicular biopsy: Mandatory in all cases of azoospermia.** Diagnostic. Therapeutic in cases with ICSI planned [Intracytoplasmic sperm injection]
- **Vasography** for looking for the obstruction only after biopsy
- **USG** for varicocele

**Management**

<b>Pretesticular cause</b>	<ul style="list-style-type: none"> <li>• Correction of the underlying cause</li> </ul>
<b>Varicocele</b>	<ul style="list-style-type: none"> <li>• Surgical correction</li> </ul>
<b>Obstructive cause with normal semen volume but azoospermia [Normal FSH]</b>	<ul style="list-style-type: none"> <li>• Do a testicular biopsy.</li> <li>• If testicular biopsy is normal, than do vasovasostomy or vasoepididymostomy to relieve the obstruction</li> <li>• If testicular biopsy is abnormal, than manage as a case of testicular failure.</li> </ul>
<b>Primary testicular failure with normal semen volume and azoospermia [Raised FSH]</b>	<ul style="list-style-type: none"> <li>• Artificial insemination with donor sperm into cervix &gt; vagina</li> <li>• Testicular sperm extraction [TESE] with intracytoplasmic sperm insertion [ICSI] followed by <i>in-vitro</i> fertilisation and implantation</li> <li>• Adoption</li> </ul>
<b>Low semen volume due to vasal agenesis and azoospermia</b>	<ul style="list-style-type: none"> <li>• Artificial insemination with donor sperm into cervix &gt; vagina</li> <li>• Percutaneous epididymal sperm aspiration [PESA] with intracytoplasmic sperm insertion [ICSI] followed by in-vitro fertilisation and implantation</li> <li>• Adoption</li> </ul>
<b>Low volume semen due to ejaculatory duct obstruction</b>	<ul style="list-style-type: none"> <li>• Evaluated using transrectal ultrasound</li> <li>• Also low fructose in semen and normal sperm count in seminal aspirate suggests this situation</li> <li>• Manage with transurethral resection of ejaculatory ducts [TUREJD]</li> </ul>
<b>Low volume semen due to failure of emission</b>	<ul style="list-style-type: none"> <li>• Electroejaculation</li> <li>• Sympathomimetic drug therapy</li> <li>• TESE with ICSI followed by IVF and implantation.</li> </ul>

**Q49. Write a note on development of genitourinary tract and enumerate its congenital anomalies.****Ans.**

- Intermediate mesoderm gets arranged as paired segmentally organized nephrotomes from the cervical to the sacral area
- Cervical nephrotomes are the first to develop and form the **pronephros** in 4th week of gestation which normally completely degenerate
- **Mesonephros** appears on day 24 and the nephrotomes from the 1st 3 lumbar segments persists and forms the ureteric bud whereas rest of it degenerates
- In early 5th week, the **metanephros** condensation begins in the front of sacrum, comes in contact with the ureteric bud and proliferates
- **Metanephric mesenchyme forms the nephron (Glomerulus, proximal and distal convoluted tubules and loop of Henle)**
- **Mesonephric bud forms the collecting duct, calyces, ureter and renal pelvis**
- The ascent of kidney starts at 6th week and reaches its normal location by 9th week. At this time, its primitive blood supply degenerates and new blood supply forms in the lumbar region
- If ascent does not occur—**Pelvic kidney (Complete ectopic kidney)**
- If lower poles fuse and block the ascent due to inferior mesenteric artery coming in the way—**Horse shoe kidney**

- Primitive cloaca is the distal hindgut and is the structure from which final urinary and anorectal components arise
- Similarly between 4th to 6th weeks, the urorectal septum and the lateral cloacal folds divide the cloaca into anterior urogenital sinus and the posterior cloaca
- The entry of mesonephric duct (ureter) into primitive urogenital sinus distinguishes the cephalic vesicourethral canal from the caudal urogenital sinus
- **Vesicourethral canal = urinary bladder and pelvic urethra**
- **Caudal urogenital sinus = phallic urethra, prostate and bulbourethral glands of cowper**
- **Mesonephric duct = vas deferens, seminal vesicles, epididymis**
- **Paramesonephric duct remnants = appendix of testes and utricle of prostate**
- **Mesonephric duct remnants = appendix of epididymis and paradidymis**
- The lateral cloacal folds form urogenital folds, labioscrotal folds and anal folds from anterior to posterior. The anterior end of urogenital folds fuse to form the genital tubercles
- These genital tubercles together with the rest of the urogenital folds form the lower external genitalia
- **The labioscrotal fold is the future scrotum, the genital tubercle elongation and fused urogenital folds form the glans and penile shaft**
- A groove appears in the anterior aspect of the genital tubercle, forms the urethral plate, urethral folds and finally the penile urethra
- **The most distal part of the penile urethra is formed by invagination of the surface epithelial tag.**

**The congenital anomalies that can appear during this development are as follows:**

<b>Kidney</b>	<ul style="list-style-type: none"> <li>• Agenesis</li> <li>• Ectopic kidney (pelvic, horse shoe, crossed fused)</li> <li>• Duplication of renal pelvis</li> <li>• Duplex kidney</li> <li>• Aberrant renal vessels (supernumerary renal vessels/double renal artery, left renal collar)</li> <li>• Cystic kidney disease</li> <li>• Persistent fetal lobulation</li> <li>• Medullary sponge kidney</li> </ul>
<b>Ureter</b>	<ul style="list-style-type: none"> <li>• Duplication</li> <li>• Megaureter</li> <li>• Ureterocele</li> <li>• Right retrocaval ureter</li> <li>• Ectopic ureter</li> <li>• Congenital hydronephrosis</li> </ul>
<b>Urinary bladder</b>	<ul style="list-style-type: none"> <li>• Bladder exstrophy</li> <li>• Vesicourethral reflux</li> </ul>
<b>Urethra</b>	<ul style="list-style-type: none"> <li>• Hypospadias</li> <li>• Epispadias</li> <li>• Posterior urethral valve</li> <li>• Urethral diverticula</li> </ul>

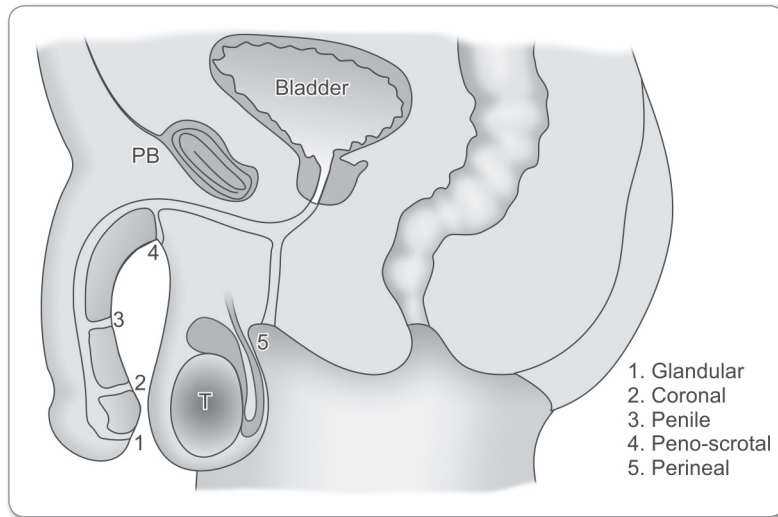
Overall, **the most common congenital anomaly of the urinary tract** is vesicourethral reflux. The most common anomaly of the upper urinary tract is duplication of the ureter.

**Q50. What is Hypospadias? Discuss its management.****Classify Hypospadias and discuss its management.****Ans.**

- Hypospadias is a congenital defect in the development of urethra and urethral folds wherein the urethra opens along the ventral surface (undersurface) of glans, penile shaft or perineum rather than its normal location at tip of glans penis
- It is the most common congenital anomaly of urethra
- Incidence: 1 in 250 cases.

**Causes**

- Embryology and embryological basis is to be written from the question on congenital development of urinary tract
- The basic defect is in fusion of the urethral folds along the undersurface (ventral surface) of penis
- Prenatal testosterone, antenatal estrogen and progesterone, IVF are all incriminated as risk factors
- Maternal use of Diethylstilbestrol is also a putative risk factor.

**Types:****Fig. 5:** Hypospadias types

From anterior to posterior are:

- First degree: Glanular, subcoronal
- Second degree: Distal penile, midshaft, proximal penile
- Third degree: Penoscrotal, scrotal, perineal.

**Clinical features:**

- External meatus opens on the undersurface of penis

- Ventral chordee
  - The absent length of urethra is replaced by a fibrous un-recanalised cord like structure which is called chordee
  - Chordee extends from the abnormal opening to the normal anatomical opening site
  - It is fibrous therefore, cannot increase in size during erection and thus leads to a curvature in penis when it becomes erect and thus produces difficulty in sexual activities as well as pain
  - Ventral Chordee occurs in hypospadias and dorsal chordee in epispadias.
- Dorsal hood of prepuce
- Meatal stenosis
- Multiple openings of urethra can be present
- Undescended testis, congenital inguinal hernias are associated.

#### **Long-term defects**

- Infertility
- Pseudo-incontinence
- Sexual problems due to chordee
- Difficulty with micturition

#### **Management**

- Optimal time for surgery: 6-12 months
- The basic elements are correction of chordee, normalization of position of the urethral opening and making use of the hooded foreskin in plastic surgical procedures to correct the defects
- Circumcision is not to be done in patients with hypospadias for the same reason
- **Surgical options**
  - Dennis – Brown technique
  - Meatal advancement and glanuloplasty (MAGPI) for coronal variety
  - Mathieu flap procedure for distal penile type
  - Thiersch-Duplay and Bracka technique for proximal penile
  - Asopa or duckett technique of flap repair of hypospadias.

## **URO-ONCOLOGY**

**Q51. Enumerate the risk factors of renal cell carcinoma. Discuss its types and clinical features.**

**Discuss the management outline of a patient with renal cell carcinoma.**

**Ans.**

#### **Risk factors**

- **Hereditary**
  - Von-Hippel-Lindau disease
  - Hereditary papillary RCC type 1 (Ch. 7, 17, Y)



- Birt-Hogg-Dube syndrome (Chromophobe cell tumor)
- Familial leiomyomatosis and RCC Type 2 (Ch. 1)
- **Environmental**
  - Tobacco consumption in any form
  - Obesity
  - Hypertension
  - Diet low in fruits and anti-oxidants
  - Radiation exposure
  - Exposure to industrial agents such as asbestos, cadmium.

### Pathological types

<b>Clear cell RCC</b>	<ul style="list-style-type: none"> <li>• Most common type</li> <li>• Associated with VHL syndrome</li> <li>• Arise from proximal convoluted tubules</li> <li>• Cell is clear as it contains glycogen and lipid</li> </ul>
<b>Papillary</b>	<ul style="list-style-type: none"> <li>• Associated with Hereditary Papillary RCC</li> <li>• Arise from distal convoluted tubule</li> <li>• Psammoma bodies are present</li> <li>• Intratumoral hemorrhage and necrosis is a feature</li> </ul>
<b>Chromophobe cell tumor</b>	<ul style="list-style-type: none"> <li>• Associated with Birt-Hogg-Dube syndrome</li> <li>• Arise from the intercalated cells of the collecting ducts</li> <li>• Cell has a plant cell appearance</li> <li>• Best prognosis</li> </ul>
<b>Collecting duct carcinoma (Bellini duct cancer)</b>	<ul style="list-style-type: none"> <li>• Rarest type</li> <li>• Arises from the collecting ducts in medulla</li> <li>• Is associated with sickle cell trait</li> <li>• Most aggressive of all types</li> </ul>

### Clinical features

- “Too late triad” of flank mass, hematuria (most common of the three symptoms) and abdominal pain is named so because presence of all the three symptoms indicate advanced disease. It is present in only around 10% cases
- Most cases present with one or more of these symptoms
- Can also present as fever, malaise, lower limb edema due to IVC compression, varicocele due to gonadal vein compression, liver failure due to Stauffer syndrome (Non metastatic hepatic dysfunction mediated by raised IL-6 that normalizes after nephrectomy)
- Paraneoplastic manifestations include raised ESR, polycythemia, hypercalcemia, hypertension and anemia.

### Investigations

- Are outlined in the question on investigation of a renal lump
- Diagnostic is CT dedicated to kidneys
- MRI is the best non-invasive and venacavogram is the best invasive investigation for IVC involvement evaluation as a serpentine malignant thrombus that can reach upto the right atrium
- **Indications of FNAC**
  - Before labelling patient inoperable and nontreatable in metastatic disease

- Doubt of secondaries in kidney
- Doubt of lymphoma
- Suspicion of renal abscess.

### AJCC TNM Staging

#### Primary tumor (T)

- **T1** limited to kidney
  - **T1a:** Tumor limited to kidney, <4 cm in size
  - **T1b:** Tumor limited to kidney, >4 cm but <7 cm
- **T2** Tumor limited to kidney, >7 cm
  - **T2a:** Tumor limited to kidney, >7 cm but not more than 10 cm
  - **T2b:** Tumor limited to kidney, >10 cm
- **T3** Tumor or tumor thrombus extension into major veins or perinephric tissue but not into ipsilateral adrenal gland or beyond Gerota's fascia
  - **T3a:** Tumor spread to renal veins
  - **T3b:** Tumor spread into infradiaphragmatic IVC
  - **T3c:** Tumor invades supradiaphragmatic IVC or wall of the IVC
- **T4** Tumor invades adrenal glands or invades beyond Gerota's fascia

#### Regional nodes (N)

- **N0:** No regional nodes
- **N1:** Nodal involvement into regional nodes

#### Distant metastasis

- **M0:** No distant metastasis
- **M1:** Distant metastasis

<b>Stage I</b>	T1 N0 M0
<b>Stage II</b>	T2 N0 M0
<b>Stage III</b>	T3 N0 M0 T1-3 N1 M0
<b>Stage IV</b>	T4/ M1 disease

### Treatment

- **Partial nephrectomy or nephron sparing surgery**
  - **Indications**
    - Done in stage I (T1) RCC
    - Patients with bilateral RCC or RCC in solitary kidney or RCC in syndromic kidneys (VHL)
    - Patients with RCC and a systemic disease likely complication of which is renal failure
  - Open or laparoscopic procedure can be done
  - However, open procedure is preferred when the tumor is near hilum, there are multiple tumors or the patient has a tumor in solitary kidney.
- **Radical nephrectomy**
  - Indications
    - In all cases of localised Stage II,III RCC

- In all cases with stauffer syndrome
- Patients with metastatic RCC planned for immunotherapy as a means of cytoreduction.
- **Traditional radical nephrectomy** is removal of kidney with intact Gerota fascia after early ligation of the renal vessels, adrenal and lymphadenectomy from crus of diaphragm to aortic bifurcation
- Now, Adrenal and lymph nodes are removed only when indicated
- **Indications of adrenal removal**
  - Extensive involvement of kidney by the tumor
  - Upper pole renal tumors adjacent to adrenal
  - Locally advanced (Stage III) tumors
- **Indications of extensive lymphadenectomy**
  - Sarcomatoid histology
  - High grade
  - Tumor more than 10 cm size
  - Stage III/IV malignancies
- **Indications of laparoscopic procedures**
  - Tumor < 13 cm
  - No local invasion
  - Limited renal vein involvement
  - Manageable lymphadenopathy
- **Management of locally unresectable or metastatic disease**
  - Surgical resection with en-bloc resection of spleen, colon, abdominal wall should be done if R0 resection is possible with such extensive resections
  - Surgical debulking
  - **Immunotherapy: Sunitinib is the drug of choice**
  - **Other options:** Interleukin-2, Interferon alfa, Sorafenib, Vinblastine, Pazopanib.
- **Management of a case with IVC thrombus**
  - Incidence: 5-10%

<b>Type I</b>	Adjacent to ostia of renal vein	Vascular isolation with satinsky clamp followed by venotomy and removal
<b>Type II</b>	Upto the Infrahepatic IVC	Sequential clamping (Caudal IVC, Contralateral renal vein, Cephalad IVC, lumbar veins) and venotomy and removal of thrombus or resection followed by grafting/reconstruction is the procedure
<b>Type III</b>	Below diaphragm	Liver mobilization followed by vascular control as above If not possible, then complete hepatic vascular isolation done with IVC clamp above liver, pringle maneuver and a venovenous bypass from IVC below to above followed by resection and graft/reconstruction of involved IVC
<b>Type IV</b>	Above diaphragm	Require cardiopulmonary bypass and hypothermic circulatory arrest with an attendant increased risk of CVA and MI

- Morbidity increases as the level of thrombus in IVC progresses higher. But, it has no bearing on mortality issues.

**Prognosis**

- Stage is the most important prognostic factor. Previously, Robson staging was used. Now, AJCC TNM staging system is being used.
- IVC wall invasion is associated with a poor prognosis.

**Q52. Write a note on risk factor for urinary bladder cancer and discuss its types. Discuss the management outline of a case of carcinoma urinary bladder.**

**Ans.**

**Risk factors**

- Squamous cell carcinoma
  - Chronic irritation of bladder due to stone, catheter or UTI
  - Schistosoma hematobium
  - Bladder diverticula
- Transitional cell cancer
  - Schistosoma hematobium
  - Smoking is the most important risk factor
  - Pelvic radiation exposure
  - Occupations such as work in chemical factories with dyes/naphthalene/hydrocarbons exposure, printing industries, rubber goods manufacturing units, leather work
  - Drugs such as cyclophosphamide, phenacetin and chlornaphazine
- Adenocarcinoma
  - Urachal remnants such as urachal cyst/ fistula
  - Ectopia vesicae.

**Pathological types**

- Transitional cell cancer (m.c. type)
- Squamous cell cancer—always to be treated with radical cystectomy
- Adenocarcinoma—apart from above mentioned risk factors, it also occurs in intestinal conduits, ureterosigmoidosomies and neobladders. Treated with radical cystectomy and pelvic lymph node dissection.

**Premalignant lesions**

- Benign papillary tumor of bladder (Kiss cancer)
- Malignant cystitis (Carcinoma—*in-situ*)

**Clinical presentation**

- Age: 6th or 7th decade
- Sex: Males
- High socio-economic status patients
- Painless hematuria is the most common presentation
- Features of UTI
- Lower urinary tract symptoms
- Metastasis: Obturator node, liver metastasis, Bony involvement.

### Investigations

- Urinary cytology
- Cystoscopy
- Transurethral resection is also included in staging investigation
- Tumor markers: Bladder tumor antigen, NMP 22
- Pelvic MRI is preferred for staging once malignancy is confirmed. CT can also be used for staging purpose
- Management depends on the stage and grade that is obtained after pathological analysis of the TUR specimen.

### AJCC Staging

#### *Primary tumor (T)*

- **Ta:** Noninvasive papillary tumor
- **Tis:** In-situ (non-invasive flat)
- **T1:** Through lamina propria into sub-epithelial connective tissues
- **T2:** Into muscularis propria
  - **T2a**—only invades inner half of the muscle
  - **T2b**—invades into outer half of the muscle
- **T3:** Invasion into perivesical tissues
  - **T3a**—microscopic extravesical invasion
  - **T3b**—macroscopic extravesical invasion
- **T4:** Direct invasion into adjacent structures
  - **T4a**—prostate, uterus, vaginal vault
  - **T4b**—pelvic side wall and / or abdominal wall

#### *Regional nodes (N)*

- **N0:** No nodal involvement
- **N1:** Single node involved < 2cm
- **N2**
  - Single node 2-5cm or
  - Multiple nodes all < 5cm
- **N3:** One or more nodes > 5cm

#### *Distant metastasis (M)*

- **M0:** No metastases
- **M1:** Metastases identified

<b>Stage I</b>	T1 N0 M0
<b>Stage II</b>	T2 N0 M0
<b>Stage III</b>	T3/T4a N0 M0
<b>Stage IV</b>	T4b N+ with any T M0 M+ With any T and N

**Treatment**

- Malignant cystitis: 2 cycles of intravesical BCG
- Low grade, nonrecurrent single lesion Ta: TUR is the treatment
- Recurrence, multicentric lesions, high grade Ta and T1:
  - TUR should be followed by intravesical chemotherapy with mitomycin C, Epirubicin or thiotepa. Mitomycin C is the preferred agent and is to be given within 6 hours of the surgery followed by clamping of foley's catheter for 1 hour after intravesical administration
  - BCG cannot be used in this setting so early. If BCG is to be used, it is used minimum 2-4 weeks after TUR to allow complete re-epithelialization after resection
    - BCG is immunotherapy and acts by eliciting a TH1 response
    - Prerequisites
      - Urine analysis to rule out infection
      - Catheterization should be nontraumatic
      - Not to be used in patients with AIDS or other immunodeficiencies or in patients with history of BCG sepsis, current UTI or incontinence and gross hematuria due to chance of absorption and BCG sepsis
      - No pre-operative antibiotic is necessary. Pyridium is used as urinary antiseptic before the procedure
    - Dose is 80 – 120 mg with 1 vial = 40 mg dissolved in 50 ml saline, administered using infant feeding tube and clamped for 2 hours after administration
    - Complications include jaundice, fever with chills (BCG sepsis), urinary irritant symptoms (dysuria, urgency and frequency).
- **T2, T3, T4 (Stage II, III, IVA):** Radical cystectomy followed by chemotherapy whenever R0 resection is possible. Chemotherapy can be MVAC (methotrexate, vinblastine, adriamycin and cisplatin), CISCA (Cyclophosphamide, cisplatin, adriamycin) or Gemcitabine + cisplatin regimes.
- For rest of the cases [**Stage IV (any T N+ and/or M+)**]**—**The management is neoadjuvant chemotherapy with MVAC (methotrexate, vinblastine, adriamycin and cisplatin) or CVM (cisplatin, vinblastine, methotrexate) followed by surgery or radiation as deemed feasible.
- **Newer agents** for advanced metastatic transitional cell cancer (**LIVP**) – larotaxel (Activity in tumors with taxane resistance), Ixabepilone (Also used in metastatic breast cancer), vinflunine and pemetrexed (Also used in non-small cell lung cancer and pleural mesothelioma).

**Q53. Discuss the risk factors for carcinoma testis and differentiate between seminoma and teratoma.**

**Write the classification of testicular tumors and discuss its management.**

**Write a note on management outline of seminoma testis.**

**Outline the management of non-seminomatous testicular tumors.**

**Ans.** They are classified as germ cell tumors and non-germ cell tumors.

**Germ cell tumors (95%, are LDH1 positive)** are as follows:

- Seminomatous germ cell tumors [m.c.]
  - These are placental alkaline phosphatase positive, gamma glutamyl transpeptidase positive and CD 117 positive, CD 30 negative
  - **It is the most common primary unilateral or bilateral tumor of testis**
  - Types include:
    - Classic seminoma
    - Anaplastic seminoma
    - Spermatocytic seminoma—It is different from the other seminomas in that it does not arise from ITGCN, it is not associated with cryptorchidism, it is never seen in mixed germ cell tumors, It is chemorefractory, It has no i12p mutation or PLAP positivity and it is never bilateral
- Non-seminomatous germ cell tumors

<b>Yolk sac tumors</b> (Orchioblastoma, Adenocarcinoma of infantile testes, endodermal sinus tumor, juvenile embryonal carcinoma)	Has presence of hyaline globules Produces AFP <b>Schiller Duval bodies</b> are seen <b>It has the best prognosis</b> <b>Most common in children upto 3 years</b>
<b>Embryonal carcinoma</b>	<b>Most undifferentiated</b> Totipotent It produces AFP and GGT It is the <b>smallest germ cell tumor</b>
<b>Teratoma</b>	Can be of mature, immature or malignant types It is chemorefractory and radioresistant <b>Most common in prepubertal age children</b>
<b>Choriocarcinoma</b> (Hurricane tumor)	<b>It is having the worst prognosis</b> It produces Beta-HCG. It spreads by hematogenous route It is associated with metastatic cerebral hemorrhage
<b>Mixed tumors</b>	<b>It is the most common type</b> Has both germ cell and non-germ cell components Managed as non-germ cell tumor

**Non-germ cell tumors are as follows:**

- Sex cord tumor (Leydig cell tumor)—bimodal age distribution. Treatment is radical orchiectomy without retroperitoneal lymph node dissection. Gynecomastia is a risk factor for leydig cell tumor
- Stromal tumors (Granulosa/Theca cell tumor) – Bimodal age distribution. Treatment is radical orchiectomy without retroperitoneal lymph node dissection
- Gonadoblastoma (Mixed components of sex cord and stromal cells)
- **Lymphoma—It is the most common testicular tumor in older patients (> 60 years age)**
- Carcinoid tumor
- Adenoma.

**Testicular carcinoma***Risk factors*

- Cryptorchidism is the most important risk factor and increases the risk in both the testis. Also, the higher the testis more is the risk of malignancy. Also, orchidopexy does not decrease the chance of malignancy. It only facilitates early recognition
- Personal history of testicular cancer
- Family history of testicular cancer in brother or identical twin
- Presence of ITGCN (Intratubular germ cell neoplasia) i.e. carcinoma *in-situ* of testes except for spermatocytic seminoma, teratoma and endodermal sinus tumor.

*Pathology and classification is as given above.*

*Clinical presentation*

- Painless gonadal swelling (**m.c.**)
- Hydrocele (10-15% cases)
- Retroperitoneal mass (lymph node) or neck node mass
- Metastases into CNS, GI, Lungs.

*Investigations*

- Tumor markers—AFP, HCG, LDH 1, GGT, PLAP. They are produced by tumor types as mentioned above
- Ultrasound scrotum—Hypoechoic mass is suggestive
- Definitive diagnoses—FNAC from neck node or retroperitoneal mass can be done if present. FNAC from testis is contraindicated. Histopathological diagnosis is obtained by high inguinal orchidectomy in all cases
- CECT abdomen and pelvis is done for lymph node detection and neck ultrasound or CT neck and chest is done in cases with suspicion of metastases or lymphadenopathy and to rule out mediastinal germ cell tumors in suspicious cases
- Metastases from right testes goes into interaortocaval nodes and from left testes goes into paraaortic nodes in the retroperitoneum.

*Management*

- After high inguinal orchidectomy and tumor marker estimation as well as the imaging studies, the patient is staged and further management is planned.

*AJCC Staging***Primary tumor (T)**

- **Tx:** Primary tumor cannot be assessed (orchidectomy not performed)
- **T0:** No evidence of primary tumor
- **Tis:** Intratubular germ cell neoplasia (carcinoma *in-situ*)
- **T1:**
  - Tumor limited to testis and epididymis
  - May invade tunica albuginea



- May not invade tunica vaginalis
- No vascular or lymphatic invasion.
- **T2:**
  - Tumor limited to testis and epididymis
  - Involvement of tunica vaginalis
  - Vascular or lymphatic invasion
- **T3:** Invasion of spermatic cord
- **T4:** Invasion of scrotum.

**Regional nodes (N)**

- **Nx:** Nodes cannot be assessed
- **N0:** No evidence of nodal involvement
- **N1:** One or more lymph nodes involved but all < 2 cm in greatest dimension
- **N2:** One or more lymph nodes involved 2–5 cm in greatest dimension
- **N3:** One or more lymph nodes involved > 5 cm in greatest dimension.

**Distant metastasis (M)**

- **Mx:** Presence of metastases cannot be assessed
- **M0:** No evidence of metastases
- **M1:** Distant metastases present
  - **M1a:** Non-regional lymph node OR pulmonary metastases
  - **M1b:** Distant metastases not fulfilling M1a

**Serum tumor markers (S)**

- **Sx:** No serum tumour markers available
- **S0:** Within normal limits
- **S1:**
  - Alpha foetoprotein : < 1000 ng/mL
  - Beta HCG : < 5000 IU/L
  - LDH : < 1.5 x upper limit of normal
- **S2**
  - Alpha fetoprotein : 1,000 – 10,000 ng/mL
  - Beta HCG : 5,000 – 50,000 IU/L
  - LDH : 1.5 – 10 x upper limit of normal
- **S3**
  - Alpha fetoprotein : > 10,000 ng/mL
  - Beta HCG : > 50,000 IU/L
  - LDH : > 10 x upper limit of normal

**Simplified staging is**

- **Stage 1** is node negative
- **Stage 2** is node positive non-metastatic disease
- **Stage 3** is metastatic disease
- **There is no stage 4** in carcinoma testes AJCC staging
- Any patient with **tumor marker status is S stage**. For example Stage IS and so on.

**Seminoma**

- Dog leg radiotherapy is the treatment consisting of administration of 25–35 Gy radiation to bilateral retroperitoneum and ipsilateral pelvic field
- It is the treatment of choice till stage IA, IB, IIA, IIB
- Stage IIC onwards and in IS, IIS—manage with chemotherapy [bleomycin, etoposide, cisplatin (BEP) 3 cycles or 4 cycles EP only].

**Nonseminoma**

- Retroperitoneal lymph node dissection (RPLND) is the procedure wherein all the lymphatic tissues between the renal vessels above, the common iliac bifurcation below and the ureters on the side are resected. Unilateral, bilateral and nerve sparing variants are available. Nerve sparing RPLND is now the preferred procedure and is performed laparoscopically also
- Nerve sparing retroperitoneal lymph node dissection (RPLND) for patients of stage IA, IB, IIA, IIB if they do not have bulky disease (> 3 cm) or very high tumor marker levels postorchiectomy
- If the patient is stage IIC onwards and in IS, IIS, or has a very bulky disease—plan is neoadjuvant chemotherapy followed by resection of residual disease or RPLND
- The only exception to this line of management is a teratoma wherein, only surgery is the treatment as it is chemorefractory and radioresistant.

**Q54. Write a note on risk factors and management of carcinoma penis.****Ans. Risk factors**

- Neonatal circumcision virtually eliminates the risk of penile carcinoma. Adult circumcision does not protect against it
- PUVA
- UV radiation
- Human papilloma virus infection – Buschke Lowenstein tumor
- Multiple sexual partners
- Tobacco/trauma/smoking
- Balanitis xerotica obliterans (lichen sclerosus).

**Pre-malignant lesions**

- Leukoplakia
- Long standing genital warts
- Cutaneous horn
- Benign variant of basal cell cancer of penis is called **epithelioma of pinkus**

**In-situ lesions**

- Erythroplasia of Queyrat on glans and prepuce—red, velvety, well marginated lesion which may ulcerate
- Bowen's disease on penile shaft/genitalia/perineum.

**Preventive vaccines**

- Gardasil (HPV 6, 11, 16, 18)
- Cervarix (HPV 16, 18)

**Most common site** = Glans > Prepuce.

### Pathology

- Exophytic or papillary group
- Flat or ulcerative lesion
- Squamous cell cancer or basal cell cancer.

**Lymphatic drainage from penis** is bilateral and goes from superficial inguinal lymph nodes to deep inguinal nodes to pelvic lymph nodes (external iliac, internal iliac, obturator nodes).

### Clinical presentation

- Ulcerated or exophytic lesion
- Foul smelling
- Inguinal lymphadenopathy
- Hypercalcemia without detectable osseous metastases has been associated.

### Investigations

- Penile ultrasound—to know invasion into tunica albuginea
- MRI—for lesions thought to invade corpus cavernosum
- Biopsy is mandatory before initiation of any therapy
- CT of inguinal and pelvic areas are done to look for direct involvement as well as lymphadenopathy
- However, CT offers no additional advantage over physical examination, especially in the patients with no palpable adenopathy. Therefore, the gold standard for inguinal node involvement detection is physical examination.

### Management

**Older staging system**—Jackson staging.

### AJCC Staging

*Primary tumor (T)*

- **TX**: Primary tumor cannot be assessed
- **T0**: No evidence of primary tumor
- **Tis**: Carcinoma *in-situ* (cancer that is only in the top layers of skin). This is sometimes called erythroplasia of Queyrat when it occurs on the glans of the penis. It can be called Bowen's disease when it occurs on the shaft of the penis
- **Ta**: Verrucous (wart-like) carcinoma that is only in the top layers of skin (non-invasive)
- **T1**: Tumor has grown into the tissue below the top layers of skin (called the subepithelial connective tissue)
  - **T1a**: The cancer has grown into the subepithelial connective tissue, but it has not grown into blood or lymph vessels. The cancer is grade 1 or 2
  - **T1b**: The cancer has grown into the subepithelial connective tissue and either it has grown into blood and lymph vessels OR it is high-grade (grade 3 or 4)

- **T2:** Tumor has grown into one of the internal chambers of the penis (the corpus spongiosum or corpora cavernosum)
- **T3:** Tumor has grown into the urethra (the tube that carries urine and semen outside the body)
- **T4:** Tumor has grown into the prostate or other nearby structures N categories

#### *Regional nodes (N)*

- **NX:** Nearby lymph nodes cannot be assessed
- **N0:** No spread to nearby lymph nodes
- **N1:** The cancer has spread to a single lymph node in the groin (inguinal lymph node)
- **N2:** The cancer has spread to more than 1 inguinal lymph node
- **N3:** The cancer has spread to lymph nodes in the pelvis and/or the cancer in the lymph nodes has grown through the outer covering of the lymph node and into the surrounding tissue

#### **Metastases (M)**

- **M0:** The cancer has not spread to distant organs or tissues
- **M1:** The cancer has spread to distant organs or tissues (such as lymph nodes outside of the pelvis, lungs, or liver)

<b>Stage 0</b>	Tis N0 M0 Ta N0 M0
<b>Stage I</b>	T1a N0 M0
<b>Stage II</b>	T1b N0 M0 T2 N0 M0 T3 N0 M0
<b>Stage III A</b>	T1-3 with N1 disease
<b>Stage III B</b>	T1-3 with N2 disease
<b>Stage IV</b>	T4 with Any N N3 with any T M0 M+ With any T and N

#### **Tis/Ta/T1 grade 1 or 2 lesions**

- Organ sparing or glans sparing procedures
- Topical treatment with 5-Fluorouracil/Imiquimod for Tis
- Mohs micrographic surgery
- Radiation therapy
- Laser/limited excision strategy
- Circumcision/glans removal with shaft sparing strategies.

#### **Standard treatment protocol**

- Glans/distal shaft—partial penectomy with 2 cm margin
- Proximal penis/2 cm margin not possible—total penectomy with perineal urethrostomy.

#### **Management of lymphadenopathy**

- **SLNB** (Sentinal lymph node biopsy) in carcinoma penis is called **CABANA procedure**

<b>Traditional indications of superficial inguinal lymph node dissection</b>	<ul style="list-style-type: none"> <li>• Persistent adenopathy after treatment of primary and 4-6 weeks of antibiotics to rule out infection as the cause of adenopathy in biopsy negative cases</li> <li>• FNAC or Excision biopsy positive for metastasis</li> <li>• New lymphadenopathy during follow up</li> <li>• All fit patients with pT2 or greater or grade 3 or more, or vascular invasion on histopathology</li> </ul>
<b>Bilateral superficial inguinal lymph node dissection</b>	<ul style="list-style-type: none"> <li>• For patients presenting with unilateral positive adenopathy at presentation</li> <li>• Not for patients presenting with unilateral positive adenopathy after primary is treated</li> </ul>
<b>Pelvic lymphadenopathy (Deep ilio-inguinal lymph node dissection)</b>	<ul style="list-style-type: none"> <li>• When inguinal node is metastatic with extracapsular spread on pathology report</li> <li>• Most patients with superficial inguinal node biopsy positive</li> </ul>

#### **Indications of adjuvant chemotherapy**

- N2,N3 lesions
- More than 2 positive lymph nodes on biopsy.

**Radiotherapy** is used in these indications when chemotherapy is contraindicated.

#### **Indications of neoadjuvant chemotherapy**

- Bilateral FNAC positive lymph nodes < 4 cm
- Fixed nodal metastasis
- Mobile nodes 4 cm or greater in size/pelvic nodes positive on preoperative imaging.

**Most important prognostic factor** is the lymph node status.

**Q55. Enumerate the risk factors for carcinoma prostate. Write a note on medical management of carcinoma prostate.**

**Outline the management of carcinoma prostate.**

**Ans.**

- It is the most common cancer of males.

#### **Risk factors**

- Advancing age
- Deficiency of anti-oxidants in diet and high fat intake in diet
- Mutation-GSTP-1 (Ch.11)
- Racial predisposition to African American men.

#### **Pathology**

- It is adenocarcinoma
- The neoplasm contains smaller glands with no branching. The glands are closely packed and lack the basal cell layer in its epithelial lining
- A gleason score of 7 or more in a biopsy specimen suggest an aggressive malignancy
- Spread can be hematogenous to lungs and lumbar vertebra, lymphatic to obturator nodes and direct invasion into urinary bladder or other nearby viscera.

**Clinical presentation**

- Present as urinary retention
- Present with symptomatic metastases
- DRE reveals hard, nodular prostate with irregular surface and obliteration of median sulcus with induration.

**Investigations**

- TRUS + PSA is the screening technique of choice
- MRI pelvis is the investigation of choice and can be performed from surface or with help of endorectal coil
- CT is done for bony and lymphatic metastasis
- Indications of bone scan
  - Advanced stage disease (T3b, T4)
  - Elevated PSA level ( $>$  or  $=$  15 ng/ml)
- Blood investigations may reveal anemia, deranged kidney function tests, elevated PSA and elevated alkaline phosphatase suggestive of bony metastasis
- Tumor markers
  - PSA
  - ALP
  - Prostatic acid phosphatase
  - Alpha methyl CoA racemase.

**AJCC Staging***Primary tumor (T)*

- T1: Not palpable via DRE or seen using TRUS
  - T1a—cancer found incidentally during TURP—less than 5% of the gland
  - T1b—cancer found incidentally but over 5% of the gland is involved
  - T1c—found by needle biopsy for a raised PSA
- T2: Palpable on DRE, but confined to the prostate
  - T2a—less than half of one side of the gland
  - T2b—more than half of one side
  - T2c—cancer on both sides of the prostate
- T3: Spread outside the prostate
  - T3a - tumour has extended outside of the prostate on one side.
  - T3b - tumour has extended outside of the prostate on both sides.
  - T3c - tumour has invaded one or both of the seminal vesicles, which are small bag-like organs near the bladder
- T4: Spread into the adjacent tissues
  - e.g. bladder sphincter, rectum or pelvic side wall.

*Nodal status (N)*

On CT a 1 cm short axis diameter cut off is used if it is purely on size grounds.

- **N0:** No spread to lymph nodes
- **N1:** One or more nearby lymph nodes involved.

*Metastases (M)*

- **M0:** No spread beyond regional lymph nodes
- **M1:** Spread beyond local nodes
  - **M1a**—distant lymph nodes outside the pelvic
  - **M1b**—bony metastases
  - **M1c**—other organ involvement independent of bony involvement
    - e.g. lungs, liver, brain.

**Treatment**

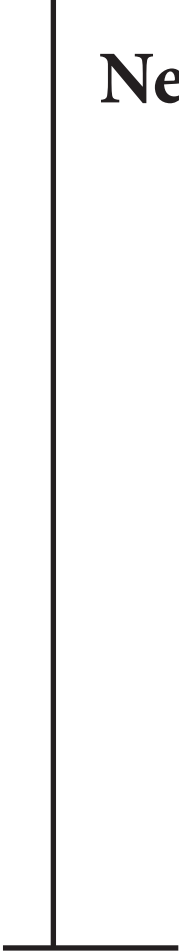
- Surgery is advised only in patients with T1b, T1c, T2 tumors and good performance status with young or middle age group
- For T1a—plan is follow up with DRE + PSA
- For all other patients with non-metastatic disease, plan is radiotherapy alone or watchful waiting
- For metastatic disease, hormonal ablation therapy with LHRH and flutamide or bilateral orchiectomy and flutamide is the treatment of choice
- Drug therapy in refractory metastatic prostate cancer—Sipuleucel T and Cabazitaxel.

# SECTION

# 4



**Neurosurgery**

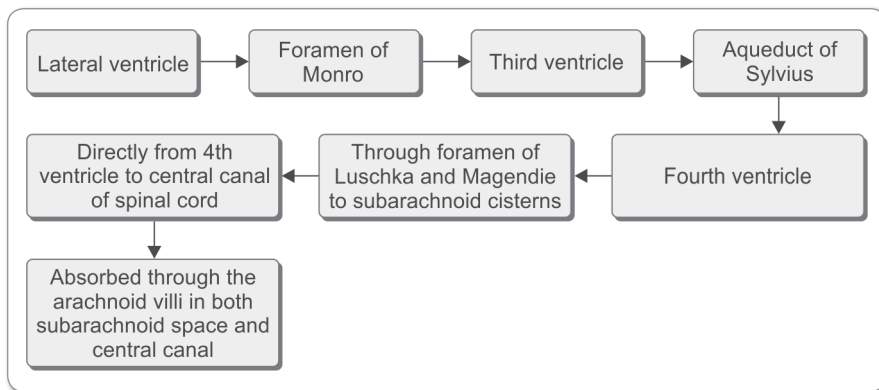






**Q1. Write a note on physiology of CSF and its circulation.****Write a note on pathway of CSF in the brain.****Ans.**

- Normal volume—150 mL
- Production rate—20 mL/h, active process by choroid plexus
- Absorption is at arachnoid villi and is pressure dependent
- Relative to plasma, it has low calcium and potassium and high magnesium, chloride, lactate and phosphate
- pH = 7.33–7.35.

**Pathway****Q2. What is hydrocephalus? What are its types?****Discuss the clinical features and management of hydrocephalus.****Ans.****Definition**

Hydrocephalus is a condition in which there is disequilibrium between CSF production and absorption, leading to raised ICP, and is often associated with dilated ventricles.

However, it is important to remember that not all patients with ventriculomegaly have hydrocephalus and not all patients with hydrocephalus necessarily have enlarged ventricles.

**Incidence**

- Ranges from 0.2–3.5/1000 birth
- Higher incidence in the offspring of elderly primi.

**Etiology of hydrocephalus**

The etiology of hydrocephalus depending on age group of patients:

- Premature infants:** Posthemorrhagic hydrocephalus leads to fibrosing arachnoiditis, meningeal fibrosis and subependymal gliosis altering the physiology of CSF flow.
- Full-term infant:** Aqueductal stenosis, Dandy-Walker malformation, tumors, arachnoid cyst, vein of Galen malformation, Chiari malformation and intrauterine infection.

- c. **Older children:** Tumors, trauma and infection can lead to hydrocephalus due to obstruction of CSF flow along its ventricular pathway.

### Also classified as

#### *Congenital*

- Aqueductal obstruction/stenosis—CSF flow is impaired when cross sectional area of aqueduct  $< 0.25$  mm
- Chiari malformation type 2 with/without associated myelomeningocele
- Chiari type 1 malformation may occur with associated aqueductal obstruction/stenosis
- Secondary aqueductal stenosis—due to intrauterine infection and germinal matrix hemorrhage
- Dandy-walker malformation—atresia of foramen of Luschka and Magendie with dilated fourth ventricle and enlarged posterior fossa
- X-linked inheritance.

#### *Acquired*

- Infections (m/c cause of communicating HCP)—post meningitic HCP e.g. TB, purulent bacterial, cysticercosis
- Post haemorrhagic—post SAH due to trauma or aneurismal bleed, post-intraventricular hemorrhage
- Secondary to masses
  - Vascular malformations
  - Neoplastic—tumors surrounding aqueduct, e.g. medulloblastoma and colloid cyst in 3rd ventricle
- Postoperative state—after posterior fossa tumor surgery
- Neurosarcoidosis
- Constitutional ventriculomegaly
- Associated with spinal tumors.

### Classification of hydrocephalus

1. **Dandy and Blackfan:** Communicating (nonobstructive) and Noncommunicating (obstructive).

Obstructive hydrocephalus—to obstruction to the CSF flow before arachnoid granulations within the ventricular system, such as blockage at the aqueduct of Sylvius or the basal foramen of Luschka and Magendie

Communicating hydrocephalus on the other hand results due to obstruction outside the ventricular system, at the level of subarachnoid space or the arachnoid granulations.

2. **Gorvers** classified as acute and chronic hydrocephalus

Acute hydrocephalus having a rapid decompression with underlying condition and present with elevated intracranial pressure.

Chronic hydrocephalus can be due to intracranial tumors, hemorrhage, trauma and infection.

**Clinical features**

Clinical features according to age.

	Symptoms	Signs
Infants	<ul style="list-style-type: none"> <li>• Vomiting</li> <li>• Reduced activity</li> <li>• Poor feeding</li> <li>• Drowsiness</li> </ul>	<ul style="list-style-type: none"> <li>• Dilated scalp vein</li> <li>• Failure of upward gaze</li> <li>• Head enlargement</li> <li>• Sunset sign</li> <li>• Tense fontanelle</li> <li>• Increased limb tone</li> </ul>
Children	<ul style="list-style-type: none"> <li>• Headache</li> <li>• Slowing of mental capacity</li> <li>• Vision deterioration</li> <li>• Vomiting</li> <li>• Drowsiness</li> </ul>	<ul style="list-style-type: none"> <li>• Failure of upward gaze</li> <li>• Unsteady gait</li> <li>• Papilledema</li> <li>• Cracked pot sign</li> <li>• Large head</li> </ul>

An increase in head circumference by more than two centimeter in any month is a sign of progressive hydrocephalus.

In congenital hydrocephalus the infant may be normal at birth and show subsequent enlargement of head especially at the age of nine month and three year of age.

*Acute Hydrocephalus*

Headache, vomiting, nausea, respiratory difficulties (Cheyne stroke) and papilledema.

*Chronic hydrocephalus:*

Enlarged head, percussion of head produces the classic cracked pot sound (macewan sign), restricted upward gaze (perinaud syndrome), setting sun sign due to weakness of upward gaze as dilated suprapineal recess compresses the quadrigeminal plate, sutural diastasis, visible scalp vein and impaired mentation.

**Investigations:**

1. **X-ray skull:** Erosion of posterior clinoids, silver beaten appearance and ballooning of sella is the evidence of chronic raised intracranial pressure.
2. **CT scan head:** In obstructive hydrocephalus there is both lateral and third ventricle dilated. In communicating hydrocephalus there is generalized enlargement of ventricle. Presence of periventricular lucencies and absent sulci are indicator of raised intracranial pressure.  
**Evans ratio:** Ratio is more than 30 percent, which is suggestive of hydrocephalus. Ratio FH/BPD (FH—frontal horn width, and BPD—bi parietal diameter).
3. **MRI brain:** MRI scanning shows the extent of dilatation of ventricle, site, nature of block, and its resultant effect. Cine Mode MRI can show the flow of CSF via aqueduct and is helpful in establishing aqueductal stenosis. Time-spatial labelling inversion pulse (SLIP) sequence more informative for the same.
4. **Ultrasonography:** Ultrasonography through anterior fontanelle usefully demonstrate ventricular enlargement in infants. It is procedure of choice for imaging fetal brain.

**Treatment of hydrocephalus:***Prophylaxis*

A single tablet of multivitamin including 0.8 mg of folic acid, given for at least one month before conception and two months after conception was found to achieve the reducing incidence of hydrocephalus.

*Acetazolamide (Diamox)*

About 25 mg/kg/day divided dose can be increased upto 100 mg/kg/day with a strict watch on electrolytes and ABG for noncommunicating hydrocephalus, TBM and as temporary relief before surgical intervention.

*Frusemide*

0.5 mg/kg and mannitol 0.25–0.5mg/kg q 6 hourly to lower ICT.

*Steroids*

dexamethasone 0.25–1 g/kg/day can be recommended for a short time in post-infective hydrocephalus.

**Surgical interventions:**

- **Lumbar drainage**
- **Ventriculoperitoneal shunt (VP shunt)**
  - The most accepted surgical procedure
  - The peritoneal end can be inserted in peritoneal cavity through open or laparoscopic techniques.

**Complications of shunt surgery**

- Infections—*Staphylococcus epidermidis* or *aureus* are usually involved with infant at particular risk
- Subdural hematoma, shunt obstruction
- Low pressure state
- Intestinal volvulus around the tube, ascites, hernia, hydrocele
- Slit ventricle syndrome, subdural effusion.
- **Ventriculoatrium shunt (VA shunt)**
  - Shunts ventricles through internal jugular vein to superior vena cava
  - particularly useful in the presence of abdominal disease. Success rate of VP shunt is 70–90%.
- **Torkildsen shunt**
  - Shunts ventricles to cisternal space
  - Used only for acquired HCP as congenital HCP don't have normal subarachnoid pathways.
- **Endoscopic third ventriculostomy (ETV):**
  - ETV is an excellent procedure for hydrocephalus when internal shunting is done within ventricular system
  - An osteomy is made inside the third ventricle which drain CSF in prepontine cistern by passing aqueductal obstruction
  - success rate varies from 70–90%.

**Indications**

- May be used in obstructive HCP
- Slit ventricle syndrome—due to excessive drainage of CSF post VP shunt
- In shunt infection as salvage procedure
- In recurrent VP shunt failure.

**Contraindications**

Relative contraindication is communicating HCP as CSF is not absorbed at arachnoid granulation.

**Q3. Enumerate the criteria for brain death.**

**Ans.** Brain death requires both cessation of function and irreversibility of cessation of either cardiopulmonary system or entire brain including brainstem.

**Criteria in adult****• Absence of brainstem reflexes**

- Absent light reflex
- Absent corneal reflex
- Absent oculoccephalic reflex (Doll's eye phenomenon)—contraindicated in cervical spine injury
- Absent oculovestibular reflex (cold and warm water caloric test)—normal response is COWS (cold shift to opposite side and warm to same side)—wait for 5 minutes before testing the other ear
- Absent gag reflex (oropharyngeal reflex)
- No cough reflex.

**• Apnea test**

- Used at last and only when the diagnoses of brain death is almost certain
- C/I – COPD and CHF
- Give 100% oxygen with  $\text{PaCO}_2 \geq 40$  mm Hg. Then give apnea for > 2 minutes with passive oxygen administration at 6 L/min with  $\text{PaCO}_2 > 60$  mm Hg or  $\text{PaCO}_2 > 20$  mm Hg from baseline or pH < 7.3
- Abort test if the patient breathes/ develops significant hypotension/ oxygen saturation drops below 80 %/ significant cardiac arrhythmia occurs.

**• Absent motor function**

- No response to central pain
- Decorticate/ decerebrate posturing and seizures are incompatible with brain death
- Spinal reflexes such as flexor plantar, flexor withdrawal, abdominal and cremasteric reflex are compatible with brain death.

**• Absence of**

- Hypothermia (core temperature > 90°F or 32.2°C)
- Shock (SBP  $\geq 90$  mm Hg)
- No hepatic encephalopathy/hyperosmolar coma
- No barbiturate/meprobamate/paralytics use
- Patient should not be immediate post resuscitation as shock/anoxia/atropine all can cause dilated and fixed pupil leading to misinterpretation.

- **Clinical confirmatory tests**

Usually not required but used in difficult situations

- Bedside EEG: No brainstem activity to be observed for minimum 6 hours
- Cerebral angiography: Absence of intracranial blood flow at the level of carotid bifurcation or circle of Willis
- Cerebral radionuclide angiogram: Hollow skull sign. Minimum 6 hours observation required
- Transcranial Doppler: No diastolic flow and small peaks only in early systole
- 1 ampoule of atropine does not affect the heart rate due to absent vagal tone.

*Observation period*

Irreversible condition present	Confirmatory test used	6 hours
Irreversible condition present	Confirmatory test not used	12 hours
Irreversible condition absent	Confirmatory test not used	24 hours
Anoxic injury		24 hours

**Criteria in children**

- Not applicable for the premature infant
- Observation period in the newborn at term or after term: 7 days
- Observation period in 7 days–2 months: 2 examinations and 2 EEGs 48 hours apart
- Observation period in 2 months–12 months: 2 examinations and 2 EEGs 24 hours apart
- Observation period in > 12 months - < 5 years : 12 hours if irreversible condition present and 24 hours in unclear condition.

**Q4. Write a note on types of head injury.**

**What is primary and secondary head injury? Discuss its causes.**

**Enumerate the types of head injury and discuss their pathophysiology.**

**Ans.**

**Head injury can be classified as follows:**

<b>Primary</b>	• Skull bone fracture	• Open or closed • Depressed or nondepressed
	• Diffuse axonal injury	• Rotational acceleration/ deceleration injury
	• Intracranial hemorrhages	• EDH,SDH,SAH, ICH/IPH
<b>Secondary</b>	• Systemic	• Hypoxia, Hypotension • Pyrexia, metabolic disturbance
	• Intracranial	• Intracranial hypertension • Intracranial hematoma • Hydrocephalus • Seizure

EDH – Extradural hemorrhage, SDH – Subdural hemorrhage, SAH – Subarachnoid hemorrhage, ICH/IPH – intracerebral/intraparenchymal hemorrhage.

**Pathophysiology of head injury**

- The main factor that affects the patients of head injury is the alteration in the cerebral perfusion pressure (CPP)
- Critical parameter for brain function and survival is not actually intracranial pressure but adequate cerebral blood flow which depends on the cerebral perfusion pressure
- **CPP = Mean arterial pressure (MAP) – intracranial pressure (ICP)**

Normal ICP	mm Hg
Adults	<10–15
Children	3–7
Infants	1.5–6

Normal CPP > or = 60 mm Hg. Pathophysiological changes do not occur till the CPP is < 50 mm Hg.

- Cerebral autoregulation is maintained in the range of CPP between 50–150 mm Hg
- The intracranial pressure is due to the intracranial constituents which include brain parenchyma with ECF (1400 mL), Cerebral blood volume (150 mL), and CSF (150 mL)
- **Monro–Kellie doctrine** states that the sum of all the above mentioned intracranial constituents is constant and that increase in any one of the contents must be compensated by an equal decrease in the other component
- **So, in head injury, when ICP is raised due to any cause,** the volume of CSF is decreased by egress through foramen magnum followed by decrease in the intravenous volume by egress through Internal jugular vein. If still not compensated, arterial blood flow is decreased which leads to decrease in CPP
- If ICP continues to rise inspite of these autoregulatory measures, than herniation of brain occurs especially when ICP > 20 mm Hg which occurs as follows:

Lesion	Herniation	Vessel impaired	Brainstem damage
Temporal lobe lesion	Uncal	Posterior cerebral artery	Present
Frontoparietal lesion	Subfalcine	Anterior cerebral artery	Absent
Posterior fossa lesion	Transtentorial or tonsillar herniation		Present
Diffuse hemispheric lesion	Transtentorial herniation		Present

- **Causes of raised ICP are as follows:**
  - Cerebral edema – cytotoxic edema
  - Injury induced hyperemia, seizure or hematoma (EDH, SDH, ICH, depressed skull fracture)
  - Hydrocephalus, cerebral vasospasm
  - Hypoventilation/systemic hypertension/hyponatremia
  - Severe adult respiratory distress syndrome with hypoventilation
  - Venous sinus thrombosis
- **Cushing's triad of raised ICP** is: Hypertension, bradycardia and respiratory irregularity.



**Q5. Write a note on Glasgow coma scale (GCS).****Write a note on monitoring of a head injury patient.****Enumerate the indications of CT scan in a patient with head injury.**

**Ans.** Monitoring of a head injury patient is done in two parts—clinical and intracranial pressure monitoring.

**1. Clinical monitoring****• Pupil**

- Anisocoria or recent change in size of pupil warrant investigation.
- Positive swinging flashlight test suggest relative afferent papillary defect and is useful.

**• Consciousness level**

- Evaluated using Glasgow coma scale at admission and then as and when required or at at least 2 hour intervals.

*GCS for all patients > 4 years age is as follows:*

Points	Best eye response	Best verbal response	Best motor response
6			Obeys command
5		Oriented	Localizes pain
4	Spontaneous	Confused	Withdraws to pain
3	To speech	Inappropriate	Flexor (decorticate)
2	Open to pain	Incomprehensible	Extensor (decerebrate)
1	None	None	None

*For patients < 4 years, best verbal response changes and is as follows*

**5** – Smiles, oriented to sound and follows objects, interacts.

**4** – Consolable inappropriate crying.

**3** – Inconsistently consolable crying, moaning child.

**2** – Inconsolable crying, restless child.

**1** – No response.

GCS < 8 = intubate.

*Categorization of head injury with help of GCS and patient assessment is as follows:*

Grade	GCS	Assessment
Minimal	15	No loss of consciousness No amnesia
Mild	14	
	15	Either LOC < 5 minutes or Impaired memory or Impaired alertness
Moderate	9–13	LOC ≥ 5 minutes or Focal neurologic deficit
Severe	5–8	
Critical	3 or 4	

*Fall in GCS by 2 points calls for urgent intervention.*

- Evaluate the **signs for basilar skull fracture** (battle sign, raccoon eyes, hemotympanum, otorrhea or rhinorrhea) and **neurological assessment** of the patient. Only after this assessment proceed to radiology and further monitoring as follows:
- **Urgent CT scan indications**  
**National institute for health and clinical excellence [NICE] guidelines** for indications of CT in a patient with head injury are as follows:
  - Glasgow Coma Score (GCS) < 13 at any point
  - GCS 13 or 14 at 2 hours
  - Fall in GCS by 2 or more points
  - Development of hemiparesis or Focal neurological deficit
  - Suspected open, depressed or basal skull fracture
  - Seizures
  - Persistent vomiting or > one episode of vomiting.

*Urgent CT head scan if none of the above but:*

- Age > 65 years
- Coagulopathy (e.g. on warfarin)
- Dangerous mechanism of injury (CT within 8 hours)
- Antegrade amnesia > 30 min (CT within 8 hours).

## 2. ICP monitoring

*Indications*

- Patient with severe traumatic brain injury (EDH, SDH, ICH, Large contusion) with GCS ≤ 8 after resuscitation
- Multiple system injuries with altered level of consciousness
- Normal CT findings with 2 or more of the following: Age > 40 years, SBP < 90 mm Hg, Decerebrate/decorticate posturing
- Acute permanent liver failure with an INR > 1.5 and coma.

*Types of available monitors*

- Intraventricular catheter [external ventricular drain]
- Intraparenchymal monitor
- Subarachnoid/ subdural/ epidural screw.

**Contraindications:** Awake patient or patient with DIC and coagulopathy

**Duration of monitoring:** Discontinue monitoring after 48–72 hours of stopping mannitol.

**Complications:** Infection, intraventricular hemorrhage, epidural or subdural hematoma, malposition of catheter, obstruction of drain lumen

*Other methods of neuromonitoring*

- Jugular venous oxygen saturation ( $\text{SjvO}_2$ ) ≤ 50% suggests ischemia
- Jugular vein oxygen content
- Arterial – jugular venous oxygen content difference [ $\text{AVDO}_2$ ] – value >9 suggests global ischemia and value < 4 suggests cerebral hyperemia

- Brain tissue oxygen tension, regional blood flow monitoring and cerebral microdialysis with evaluation of lactate/pyruvate/glucose/glutamate/urea/electrolytes and calcium are other methods.

**Q6. Explain the management of a patient with head injury.**

**Outline the management of a patient with raised ICP.**

**Always remember:** Craniotomy means creating a bone flap by using burr hole elevation and replacement of the flap at the completion of surgery whereas craniectomy means removal of bone flap and placement in anterior abdominal wall or bone bank to be used for cranioplasty at a later date.

**Ans.**

**Minimal and mild head injury**

- These are patients with no symptoms or only mild headache/dizziness
- Symptomatic treatment will suffice for these patients
- Scalp hematoma, laceration, contusion or abrasion needs dressing and suturing on outpatient basis.

**Moderate head injury**

- Hospital admission and NCCT needed
- MC injury is contusion and management is as for severe head injury
- Exceptions that can have home treatment are: GCS  $\geq 14$ , no seizure or drug intoxication, intact neurology, sober adult with good access to hospital.

**Severe head injury**

- Patient present with depressed consciousness level not due to drugs/metabolic abnormalities or alcohol, focal neurological deficit, deteriorating consciousness, penetrating head injury or depressed fracture
- Management outline includes 5 main measures.

**1. Initial resuscitation and ICP monitor insertion**

*Based on ATLS protocol*

- A** – Airway with cervical spine protection
- B** – Breathing
- C** – Circulation
- D** – Disability assessment
- E** – Exposure of all parts and assessment of injury

*Indications of elective intubation*

- GCS  $< 8$
- Need of hyperventilation to decrease ICP
- Associated severe maxillofacial trauma
- Need of pharmacological paralysis for evaluation or management.

*Burr hole drainage*

Exploratory burr hole is used only when there is sudden drop of GCS/ one pupil dilated or fixed/ decerebrate posture/ needing emergency surgery for systemic injuries with nonavailability of NCCT or when NCCT is not possible due to hemodynamic instability.

*Choice of side for initial Burr hole*

- Ipsilateral to dilated pupil
- If both are dilated, than use side of the first dilated pupil
- If still in doubt, put in side of external trauma
- If no clue, place hole on left side.

*Location of Burr hole*

First temporal burr hole – 1 cm anterior to tragus and 1 cm above zygomatic arch

If no epidural, rule out subdural. If no hematoma, next perform contralateral temporal burr hole followed by ipsilateral frontal f/b ipsilateral parietal f/b ipsilateral occipital region.

**Complications:** Meningitis, brain abscess, osteomyelitis, frontal nerve/ temporal artery injury.

**2. Management of raised ICP.**

Treatment should be initiated at ICP > 20 mm Hg and CPP < 50 mm Hg.

Steps of treatment should be used in the sequence till ICP normalizes as follows:

<b>Step 1</b>	Intubation, normocarbic ventilation (PaCO <sub>2</sub> 35–40 mm Hg), adequate analgesia with/out neuromuscular paralysis (e.g. Vecuronium) and sedation
<b>Step 2</b>	Elevate head end of bed by 30° with tight tape stabilizing neck
<b>Step 3</b>	Ventriculostomy or external ventricular drainage of 3–5 mL CSF
<b>Step 4</b>	<ul style="list-style-type: none"> <li>• Mannitol 1g/kg over 30 minutes bolus f/b 0.25–5 g/kg IV 6 hourly or 10–20 mL, 23.4% saline bolus (plasma expansion reduces blood viscosity and increases blood supply to brain and increased serum tonicity decreases cerebral edema)</li> <li>• Caution as disruption of blood brain barrier leads to vasogenic edema/ acute renal failure/combination of corticosteroids + phenytoin + mannitol leads to hyperosmolar nonketotic coma</li> <li>• Hypertonic saline is not used routinely</li> <li>• Loop diuretics act synergistically with mannitol to reduce cerebral edema</li> </ul>
<b>Step 5</b>	Mild to moderate hypothermia without shivering and hyperventilation for 24 hours [to PaCO <sub>2</sub> > 25 mm Hg]. Decreasing CO <sub>2</sub> causes cerebral vasoconstriction and decreases cerebral blood flow and helps when used only after 24 hours at least and after 5 days is best time to use hyperventilation
<b>Step 6</b>	Decompressive craniectomy (removal of a bone flap at least 12 cm diameter with associated frontal or temporal contusectomy and duroplasty)
<b>Step 7</b>	Eisenberg protocol: Barbiturate (pentobarbital/ thiopental/propofol) coma under EEG control [90% burst suppression] and vasopressors to maintain CPP if necessary
<b>STEP</b>	At any time if signs of raised ICP increase – do NCCT f/b surgery

**3. Management of seizure.***Prophylactic role*

**There are two categories of post-traumatic seizures: Early < 7 days and late > 7 days**

Anticonvulsants may be used for prevention of early seizures but never for late post-traumatic seizures.

*Indications of anticonvulsant therapy after trauma*

- Acute SDH, EDH, ICH

- Open depressed skull fracture
- Penetrating head injury
- Seizure within 1st 24 hours
- GCS < 10
- Cortical hemorrhagic contusion on CT.

Phenytoin is the drug of choice and is to be continued for minimum 6 months before discontinuation.

*Management of acute seizure*

- Lorazepam 0.1mg/kg IV at 2mL/min f/b
- Phenytoin 20 mg/kg IV bolus and 5 mg/kg in three divided doses f/b
- Phenobarbital 20 mg/kg IV bolus and additional 5–10 mg/kg f/b
- Anesthesia with midazolam/propofol/pentobarbital.

**4. General patient care**

- Skin/ eye/ back/ limb care
- DVT prophylaxis
- Nutrition and bed sore prevention
- Antibiotics and antacids (proton pump inhibitors) to prevent infection and stress ulcers
- Tetanus prophylaxis and gas gangrene prophylaxis as needed.

**5. Surgical management of intracranial injuries–**

EDH/SDH/ICH as discussed in the next questions.

*Indications for surgery in ICH/IPH*

- Volume > 50 cm<sup>3</sup>
- Midline shift > 5 mm
- Basal cistern compression
- Clot volume > 20 cm<sup>3</sup> with neurological deterioration.

*Indications of surgery in depressed skull fracture*

- Associated underlying parenchymal deficit
- Depression > 10 mm or greater than calvarial thickness
- Spicules which can tear dura
- Open depressed fracture with CSF leak.

**Q7. Write a note on epidural hematomas (EDH).**

**Discuss the management of a patient with extradural hematoma.**

**Ans.**

**Definition**

It is the collection of blood or blood products between skull bone and dura.

- **Incidence:** 1% of head trauma patients
- **M:F** = 4:1
- Is more common in young adults and rare in age < 2 years and > 60 years.

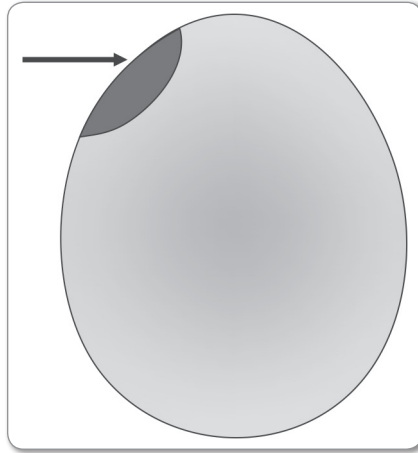


Fig. 1: Epidural hematomas

### Source of bleeding

- Arterial: Anterior branch of middle meningeal artery is the source in 85% cases
- Middle meningeal veins and dural sinuses
- Fractured bones and fragments
- Tumor or arteriovenous malformation bleeds
- Postoperative EDH – after VP shunt surgery.

### Site

70% occur over the hemispheres at pterion f/b frontal > occipital > posterior fossa.

### Clinical features

- Brief loss of consciousness f/b lucid interval of several hours f/b obtundation, contralateral hemiparesis and ipsilateral pupillary dilatation
- Headache, nausea, vomiting
- Seizures and memory impairment
- **Kernohan's notch phenomenon:** Shift of the brainstem away from the mass may produce ipsilateral hemiparesis due to compression of the opposite cerebral peduncle on tentorial notch. These patients also have ipsilateral pupil dilated.

### Investigations

- **Noncontrast CT head:** Biconvex (Lenticular) shape hyperdense area adjacent to skull. Can be crescent shaped in 5% cases. EDH can cross Falx cerebri but is usually limited by skull sutures, and dural attachments
- Routine preanesthetic checkup in anticipation of urgent surgery.

### Treatment

- Manage as per the management outline given in previous question on head injury. Remember that mannitol is contraindicated here
- Observation for all patients with stable GCS when they do not fit the criteria for urgent surgery

- **Indications for urgent surgery (craniotomy) are:**
  - Volume  $> 30 \text{ cm}^3$
  - Thickness  $> 1.5 \text{ cm}$
  - GCS  $< 8$
  - Focal neurological deficit, pupillary changes and new onset seizure also merit consideration for surgical management
  - Volume  $> 30 \text{ cm}^3$  + Thickness  $> 1.5 \text{ cm}$  + midline shift  $> 5 \text{ mm}$  is also an indication
  - Any deterioration in GCS [increasing drowsiness, pupillary changes. Hemiparesis, new onset seizure] prompt surgical intervention
- Trauma flap and craniotomy with evacuation of the clot are used to remove the epidural hematoma. Brain pulsation should return after decompression
- Mortality – 20-25% and is more so in the patients without lucid interval. Death is usually due to uncal herniation causing injury to midbrain
- Bilateral Babinski positive or decerebration in preoperative period has a poor prognosis.

**Delayed epidural hemorrhage** is when a normal brain CT within 6 hours of trauma is followed up to reveal an epidural hematoma in the new CT. This can occur in patients of head injury treated for head injury by rapid decrease in ICP medically (mannitol/lasix) or surgically (evacuating contralateral hematoma) or in patients with coagulopathy or skull fracture.

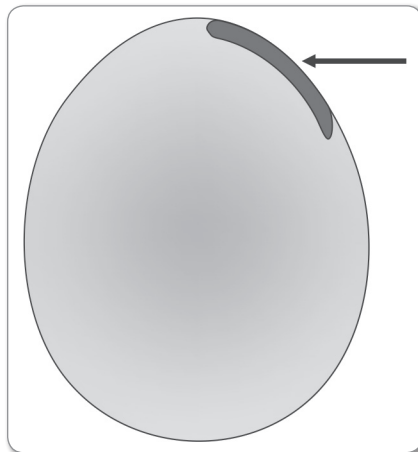
**Posterior fossa EDH** occurs due to dural sinus tears [transverse sinus, sigmoid sinus, occipital sinus]. Treatment is by suboccipital craniectomy for symptomatic lesions with mass effects and observation for all other lesions.

**Q8. Write a note on subdural hematoma (SDH).**

**Discuss the management of chronic subdural hematoma.**

**Ans.** It is collection of blood or blood products between the dura and arachnoid layer which occur as a result of arterial or venous hemorrhage.

**Classically SDH is due to tearing of the bridging veins** that span the subdural space draining the cortical blood to venous sinus.



**Fig. 2:** Subdural hematoma

**Classification**

SDH	Pathology	Clinical
Acute	Clot and blood of 48 hours	Within 3 days
Subacute	Clot and fluid (2–14 days)	3 – 21 days
Chronic	Fluid (> 14 days)	> 21 days

**Acute SDH (50–60% cases)***Causes*

- Tear in bridging veins from acceleration – deceleration injury during head trauma
- Accumulation around the parenchymal lacerations due to injury to pial arterioles (burst temporal lobe m.c. cause).

*Clinical features*

- Usually no lucid interval and neurological deficits are late to occur
- Range from mild as headache, nausea, limited verbal response, forgetfulness to severe as loss of consciousness, recurrent vomiting or severe headache, focal neurological deficits, seizures, ENT bleed, anterograde or retrograde amnesia.

*Investigations*

- **Plain CT brain:** Crescentic hyperdense mass at convexities of the brain that do not cross falx cerebri but can cross sutures with or without midline shift
- Density of SDH changes with time, **acute is hyperdense, subacute is isodense and chronic is hypodense**
- Routine preanesthetic checkup in anticipation of urgent surgery
- **MRI:** Gives the type of SDH definitively.

*Treatment*

- **Indications of surgery are as follows:**
  - Acute SDH with thickness > 10 mm or midline shift > 5 mm
  - Acute SDH with thickness < 10 mm and midline shift < 5 mm but with GCS drop by 2 or more points after admission or asymmetric pupils or dilated and fixed pupils or ICP > 20 mm Hg
- **Operate as soon as possible:** > 4 hours delay increases mortality
- Trauma flap and craniotomy with U shaped dural incision and evacuation of the clot are used to remove the subdural hematoma. Brain pulsation should return after decompression
- Mortality—50-90% due to associated brain injury.

**Intersphincteric subdural hematoma**

- Hematoma along the falx between the two cerebral hemispheres is one of the indicators of child abuse or an indication of head trauma or ruptured aneurysm in adults
- May be asymptomatic or present with “Falx syndrome”—hemiparesis and seizures contralateral to hematoma
- Only symptomatic lesions need to be operated as above.



**Delayed acute SDH**

- Acute SDH that is not present on initial study [CT or MRI] but presents on subsequent study
- Management is as for acute SDH.

**Chronic SDH**

- It is collection of the blood or blood products between the two layers of dura
- M:F = 3:2
- **Risk factors:** Trivial repeated head trauma, alcohol, seizures, coagulopathy, CSF shunts
- It may be bilateral (25% cases)
- Starts as acute SDH which evokes an inflammatory response, produces neomembrane by fibroblast induction which in turn is followed by increased enzymatic fibrinolysis as well as liquefaction of blood clot with consumption of factors 2,5,7,8,10 and this lead to impaired hemostasis
- **Clinical features** include symptoms from mild to severe as above
- **Investigations:** **CT** reveals sickle shaped hypodense collection with preserved underlying sulci and gyri and compensated midline shift. Lesion of variable densities are not uncommon though  
**MRI :** It is the preferred modality in children and elderly and shows hyperintense collection in both T1 and T2 images

- **Management**

- **Seizure prophylaxis**
- **Reverse coagulopathy**
- **Surgery**—for symptomatic lesions and for lesions thickness > 1cm
- **Techniques**—**Twist hole drainage** (for diameter < 5 mm), **Burr hole drainage** (for diameter of 5–30 mm) or **craniotomy** (for >30 mm diameter)
- Resolution in clinical symptoms occurs when about 20% of the collection is drained. Complete radiologic resolution require about 6 months
- Mortality—0–8%
- Complications: Seizures, intracerebral hemorrhage, recurrence, tension pneumoencephalitis, subdural empyema.

**Q9. What is spina bifida? What are its types?****Discuss the management of a patient with spina bifida.****Enumerate congenital neural tube defects and discuss spina bifida.****Ans.****Classification of congenital neural tube defects is as follows:**

Spinal dysraphism (Spina bifida)	Aperta	Meningocele Myelomeningocele (m.c.)—associated with folate deficiency, Chiari II malformation, hydrocephalus. (treat hydrocephalus first)
	Occulta	Teethered cord Dermal sinus Diastometomyelia Lipomyelomeningocele

*Contd...*

Contd...

Cranial dysraphism	Encephalocele Meningocele Dermal sinus	
Craniosynostosis	Genetic	Crouzon, Apert and Pfeiffer syndrome
	Sporadic	Sagittal (m.c.) Coronal/metopic/lambdoid/ kleeblattschadel
Chiari malformation	I – no spina bifida II – associated with spina bifida III – associated with cervical encephalocele	

### Spina bifida occulta

- Congenital absence of spinous process and variable amount of lamina without visible exposure of meninges or neural tissue
- F:M = 2:1
- Occurs because of failure of neural tube closure. Defective secondary neurulation to be specific
- Usually asymptomatic or mild pain at the dysraphic site
- Teethered cord and dermal sinus can present with split cord malformations
- MRI spine is the investigation of choice
- Usually no treatment is required except when symptomatic dermal sinus or teethered cord necessitate definitive surgical treatment.

### Spina Bifida Aperta (Meningocele and Meningomyelocele)

#### Definition:

Congenital defect in vertebral arches with cystic distension of meninges with [meningomyelocele] or without [meningocele] structural and functional abnormality of the spinal cord or cauda equina.

**Incidence:** 1–2/1000 live births. More risk if present in siblings.

Slight female preponderance.

#### Pathogenesis

Open posterior neuropore at 28 days leads to egress of CSF through the defect and development of a lake of distension of developing brain due to no connectivity between central canal and ventricles. This leads to myelomeningocele and Chiari II malformation (small posterior fossa, tonsillar herniation and hydrocephalus).

#### Risk factors

- Most accepted risk factor is folate deficiency
- Radiation exposure, tobacco smoking, anesthetic agents, lead toxicity
- Parent with neural tube defect—2–3%
- Diabetes mellitus type 1–1%
- Drugs (valproate, carbamazepine), maternal obesity, trisomy 13 and 18.

**Associated abnormality:** Chiari type II malformation, hydrocephalus, syringomyelia, polymicrogyria, defective corpus callosum.

*Clinical features*

- Mostly located in thoracolumbar or lumbar region
- Gradually enlarging swelling since birth with visible neural placode through transparent arachnoid layers
- Fluctuation and transillumination positive, nonpulsatile. Skin cover become macerated with time
- Hyperreflexia, clonus and other signs of myelopathy may be present
- Paraparesis, paraplegia and variable degree of sensory deficits may be present
- Neurogenic bladder, scoliosis, kyphosis, club foot, pulmonary hypoplasia can be present.

*Diagnoses*

- **Prenatal:**
  - Elevated **maternal serum alfa feto protein** at 16–18 weeks is 60–70% sensitive
  - **Maternal USG: Lemon sign** (scalloping of frontal sinus on a biparietal view) and **banana sign** (abnormally shaped midbrain, elongated cerebellum, obliteration of cistern magna characteristic of Chiari II malformation) – 100% sensitive
  - **Amniocentesis** and amniotic fluid acetylcholinesterase and AFP combined have 99% sensitivity
  - **MRI with fast spin echo technique:** Second line noninvasive modality
  - **Prenatal management:** Fetal surgery with intrauterine myelomeningocele repair can be attempted. Also counseling of risk in future pregnancy needs to be explained.
- **Postnatal:**
  - Clinical examination, USG, MRI catch the diagnoses in most of the cases.

*Management*

- If no other abnormalities are present, the surgery is done within 72 hours of birth
- Goal is to free the placode from the dura f/b water tight dural closure and skin closure to achieve maximum neurological function and maximum cosmetic outcome
- Avoid narcotics as these patients may have midbrain malformation which make them more susceptible to respiratory depression
- Postoperative nursing is done in prone or lateral position.

*Complications*

- Hydrocephalus—require shunt
- Syringomyelia—surgical correction
- Chiari II malformation—surgical correction
- Teethered cord and scoliosis—surgical correction.

*Outcome*

- 85% will survive and 80% will have normal IQ if managed appropriately
- However, Only 3–10% have urinary continence, the most significant long term morbidity.

**Q10. What is functional neurosurgery? Discuss its uses in brief.**

**Ans.** Functional neurosurgery is concerned with anatomic and physiologic alteration of the nervous system to achieve the desired effect with focal electrical stimulation, ablation or implantable pumps to deliver drugs to CSF or parenchyma to manage disorders such as

epilepsy, movement disorders, and treatment of chronic pain syndromes when these are refractory to conventional treatment.

### Applications

- **Deep brain stimulation**

It is placement of an electrode in particular brain site stereotactically to stimulate that area of brain

- Parkinson disease (globus pallidus interneurons, subthalamic nuclei)  
Athetosis (contralateral ventralis intermedius nucleus)  
Rigidity and bradykinesia (pallidum), and  
Hemiballismus (subthalamic nucleus)
- Epilepsy – generalized (GTCS)—centromedian nucleus of thalamus  
Partial seizure – anterior bilateral hippocampus  
Intractable seizures – left vagal nucleus stimulation
- Differentiation pain syndromes (anesthesia dolorosa, thalamic pain syndrome)- VPL or VPM nucleus of thalamus  
Nociceptive pain syndromes – stimulation of periventricular or periaqueductal gray matter
- Psychiatric disorders such as tourette syndrome, obsessive compulsive disorder, depression are also being treated.

- **Ablative procedures**

- **For spasticity** – motor point block, sciatic or obturator neurectomy, percutaneous radiofrequency foraminial rhizotomy, myelotomy or cordotomy
- **For pain—intracranial ablative procedures** such as cingulotomy, medial thalamotomy, and **spinal ablative procedures** such as cordotomy or commissural myelotomy for lateral spinothalamic tract ablation, dorsal rhizotomy, dorsal root ganglionectomy

- **Peripheral nerve procedures:** Peripheral nerve blocks, neurectomy, peripheral nerve stimulation.

### Common procedures of functional neurosurgery

- **Pallidotomy for Parkinson's disease**
- **Complex regional pain syndrome management:** Stellate ganglion block, lumbar sympathetic block, or surgical sympathectomy
- **Trigeminal neuralgia:** Microvascular decompression (procedure of choice), peripheral nerve procedures, rhizotomy of trigeminal nucleus, stereotactic ablation
- **Seizures:** Anterior temporal lobectomy, focal resections, multiple subpial resection, corpus callosotomy (for Lennox-Gestaut syndrome), hemispherectomy (for Rasmussen syndrome)
- **Sympathectomy:** Cardiac, cervical or lumbar.

### Q11. What is stereotactic surgery? Discuss in brief.

**Ans.** Stereotactic is a greek word stereo- 3 dimensional and tactic- to touch. Stereotaxis as applied to neurosurgery is concerned with the localization of a target in three-dimensional space. The target deep in the brain is not seen directly at surgery. The field has evolved using both frame-based systems and frameless systems.

**The procedure is done in two parts.**

- **First** being a CT scan or MRI (occasionally angiogram) is performed with either localizing device affixed to patient head or bony landmarks and fiducial markers to register the patients skull relative to radiographic images
- **Second** part of procedures utilizes a set of guides oriented to the same coordinate system to direct biopsy needle to target location.

**Principles of stereotactic surgery**

An object can be localized in space by using three coordinates “in front or behind, above or below and to left or right.” The coordinates are to be calculated from certain stationary points called reference points. Initially the pioneers of stereotaxy used pineal gland as the reference point. The commonly used reference points now are the anterior and posterior commissures of the third ventricle. It is better to select the reference point close to the target.

**Requisites for stereotactic surgery**

- A good stereotactic atlas
- A reliable stereotactic apparatus
- Training in stereotactic surgery.

**Stereotactic atlas**

Modern stereotactic planning system are computer based. Each brain structure is assigned a range of three coordinate numbers, which will be used for positioning the stereotactic device. In most atlases, the three dimensions are—mediolateral (x), dorsoventral (y) and rostrocaudal (z).

**Stereotactic apparatus**

- It should be possible to fix the frame rigidly to the skull
- One must be able to reapply it repeatedly in same position
- It should be possible to move the biopsy probe/electrode mm by mm in all the three dimensions in space
- The apparatus must be as light as possible and be fixed with little discomfort to the patient
- There should be provision for visualizing the electrode position at any stage during the operation
- Availability of CT/MRI compatible apparatus.

**Indications for stereotactic surgery**

- Biopsy—deeply seated cerebral lesions, brainstem lesions, multiple small lesions (AIDS)
- Catheter placement—drainage of deep lesions eg colloid cyst abscess, for intratumoral chemotherapy, radioactive implants for interstitial brachy therapy
- Electrode placement—for deep brain stimulation eg parkinsonism, chronic pain, epilepsy
- Evacuation of intracerebral haemorrhage—using urokinase or rTPA
- To localise a lesion for open craniotomy—AVM, deep tumor in eloquent brain
- Trans oral biopsy of C2 vertebral body lesions
- **Stereotactic radiosurgery using LINAC, Gammaknife or cyberknife devices.**

**Indications include:**

- Arteriovenous malformations—best for < 3 cm AVMs
- Tumors
  - Vestibular schwannomas—poor operative candidates (comorbid conditions, > 70 years), incomplete removed tumors by surgery, recurrence
  - Pituitary adenomas
  - Craniopharyngiomas
  - Pineal tumors
  - Metastasis
  - High grade gliomas
  - Meningiomas of cavernous sinus
- Functional neurosurgery—chronic pain including trigeminal neuralgia, various deep brain stimulation procedures.
- Potential- stereotactic laser surgery, foreign body removal.

**Contraindications**

- Coagulation disorders- a relative contraindication. For thrombocytopenia – PC <50000 absolute contraindication, desirable is >100000
- Inability to tolerate GA (severe comorbid conditions)
- Non cooperative for LA (psychiatric patient).

**Complications**

- Hemorrhage is most common
- Others are according to area of brain involved for lesioning.

**Q12. What is neurofibroma? Enumerate its types.****Discuss the clinical features and management of neurofibroma.****Ans.**

<b>True neuroma</b>	Neural crest	Neuroblastoma
		Chromaffinoma/pheochromocytoma
		Ganglioneuroma
	Spinal cord/ pia mater	Myelinic neuroma
<b>False neuroma</b>	Endoneurium origin	<b>Neurofibroma</b>

**Types of neurofibroma**

<b>Cutaneous neurofibromatosis</b>	<ul style="list-style-type: none"> <li>• Molluscum fibrosum</li> <li>• Scalp (Turban tumor)</li> <li>• No skin hypertrophy</li> </ul>
<b>Solitary</b>	Localized neurofibroma <ul style="list-style-type: none"> <li>• It is encapsulated round subcutaneous swelling alongside a nerve with well defined margins and mobility in direction opposite the axis of nerve</li> <li>• Can also be intramuscular, intraosseous, intracranial or in dorsal nerve root and ganglion</li> <li>• May have a history of paresthesia/pain/weakness</li> <li>• Treatment is resection with care to avoid injury to the nerve or resection with primary repair of the nerve</li> </ul>

*Contd...*

Contd...

<b>Generalized neurofibromatosis</b>	Von Recklinghausen disease <ul style="list-style-type: none"> <li>• Autosomal dominant, more common in males</li> <li>• Diagnosed when two or more of the following are met:             <ul style="list-style-type: none"> <li>– Six or more Cafe au lait spots &gt; 5 mm in prepubertal and &gt; 15 mm in postpubertal patients.</li> <li>– Two or more neurofibromas of any type or 1 plexiform neurofibroma</li> <li>– Axillary or inguinal freckle</li> <li>– Optic glioma</li> <li>– Osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with/out pseudoarthrosis</li> <li>– 2 or more lisch nodules (iris hamartomas)</li> <li>– Parent/sibling/offspring (First degree relative) with the disease</li> </ul> </li> <li>• Surgery is done for painful lumps, or lumps which cause pressure symptoms/mechanical discomfort/unsightly appearance/suspicion of malignant change</li> </ul>
<b>Plexiform neurofibroma</b>	Pachydermatocele—5th nerve most commonly involved Associated with hypertrophy and coarsening of skin
<b>Elephantiasis neurofibromatosis</b>	It is a severe form of plexiform neurofibromatosis and is a cause of elephantiasis of lower limb.
<b>Neural neurofibroma</b>	Cranial—acoustic neuroma Spinal—dumbbell tumors

**Clinical features of malignant change in neurofibroma**

- Appearance of pain
- Sudden increase in size
- Fixity
- Increased vascularity
- Signs of paralysis or anesthesia in the territory of nerve distribution.

**Q13. Give the differential diagnoses of intracranial space occupying lesions.**

**Ans.** Intracranial space occupying lesions is a broad terminology, including multitude of pathologies under single heading, with common feature of space occupying lesion in brain parenchyma, with or without mass effect on adjacent structures.

**Differential diagnosis***Infective*

- Pyogenic abscess
- Infective granulomas, e.g. tuberculomas, neurocysticercosis, histoplasmosis, etc.
- Hydatid cyst
- Fungal abscess
- Toxoplasmosis.

*Neoplastic*

- Gliomas: Low grade astrocytoma, anaplastic astrocytoma, glioblastoma multiforme, ependymomas, choroid plexus tumors, oligodendroglioma, pleomorphic xanthoastrocytoma

- Neuronal tumors: Ganglioglioma, dysembryoplastic neuroepithelial tumor (DNET)
- Meningeal tumours: Meningiomas and other meningiomatous neoplasms
- Pineal region tumours: Pinealoblastoma, pineocytoma, germ cell tumor
- Sellar and parasellar lesions: Pituitary macroadenoma, meningioma, craniopharyngioma
- Intraventricular lesions: Choroid plexus papilloma, choroid plexus carcinoma, central neurocytoma
- Infratentorial lesions: Medulloblastoma, ependymoma, pilocytic astrocytoma, hemangioblastoma, Lhermitte-Duclos disease, brain stem glioma, primitive neuroectodermal tumor (PNET)
- Hematopoietic tumors: Lymphoma, leukemia (chloroma)
- Metastasis
- Neoplasm like lesions: Epidermoid cyst, dermoid cyst, lipoma, Rathke's cleft cyst, arachnoid cyst.

#### *Demyelinating*

- Tumefactive demyelination
- Large lesions of multiple sclerosis or acute disseminated encephalomyelitis (ADEM)
- Hemorrhagic ADEM (Hurst's disease).

#### *Miscellaneous*

- Radiation necrosis
- Hemorrhagic infarct
- Cerebral contusion
- Arteriovenous malformation.



# SECTION

# 5

## **Cardiothoracic and Vascular Surgery**

- Cardiac and Thoracic Surgery
- Peripheral Arterial Disease for Lower Limb
- Peripheral Venous Diseases
- Miscellaneous Topics in CTVs



## CARDIAC AND THORACIC SURGERY

### Q1. What is empyema? Discuss its causes and management.

**Ans.** Empyema thoracis is collection of pus in the pleural cavity.

#### Causes

- **Infection**
  - Tuberculosis
  - Pneumonia
  - Lung abscess
  - Subphrenic abscess
  - Liver abscess
  - Mediastinal lymph node infection
  - Mediastinal abscess
- **Surgery**
  - Post-pneumonectomy
  - Leak from esophagogastric anastomosis
  - Needle aspiration from mediastinal lymph nodes/ pleural biopsy/ lung nodule
- **Bronchiectasis.**

#### Phases of empyema development

- **Exudative:** The pleural space contains no pus, only fluid
- **Fibropurulent:** There is fluid and infection
- **Organized:** Formation of loculi and pleural peels
- **Empyema necessitates:** When empyema spontaneously ruptures through the chest wall and forms a fistula, this is called empyema necessitates.

#### Management

##### *Principles*

- Drainage or removal of all contents in the pleural cavity
- Control of the infection and the foci of infection
- Re-expansion of the underlying lung.

##### *Management*

- **Investigate** for the extent of disease and the cause—chest X-ray, contrast enhanced computed tomography (CECT) chest, all cultures—pleural fluid, blood, urine, endobronchial as indicated, all blood investigations and investigations for anesthetic fitness
- Start appropriate **antibiotics**
- **Tube thoracocentesis** to drain the contents in the first two phases of disease
- **Intrapleural thrombolytic therapy** with streptokinase or urokinase is effective for early stages only. After the thrombolytic administration, the tube is to be clamped for 2–4 hours and then declamped. This can be done for upto 14 days once daily to completely drain the pleural contents

- **Surgical management**

- **Indication:** For cases in which the lung is trapped by nonelastic fibropurulent covering but remains expandable if the peel is removed and also the patient should be stable enough to withstand the procedure
- **Thoracoscopy and breakdown of septa** is the first attempted procedure.
- If the patient still does not improve, and has chronic empyema > 2–3 months with no response on closed tube drainage or with bronchopleural fistula, then the plan is to proceed towards formal **thoracotomy and decortications** for stable patients.
- If the patient is too unstable to withstand the thoracoscopy or thoracotomy, then plan for **open thoracostomy**, i.e. the pleural cavity is opened to exterior using “**Eloesser flap**” or “**Clagett window**” procedure
- These thoracostomy sites will remain open for nearly 4–6 months usually till patients resolve and then the closure can be done using intercostal muscle transposition and final skin closure.

**Q2. Enumerate the various chest traumas that you encounter in emergency department.**

**Q3. Write a note on pneumothorax.**

**Ans.** The chest trauma can be blunt or penetrating. The different injuries that can occur include the following:

**Life threatening injuries**

- Airway: Obstruction/injury
- Breathing: Tension or open pneumothorax, flail chest with pulmonary contusion
- Circulation: Cardiac tamponade, myocardial contusion, intrathoracic hemorrhage, air embolism, myocardial infarction
- Tracheobronchial injuries.

**Other injuries**

- Chest wall and pleural injuries
- Lung injuries
- Aortic injuries
- Esophageal injuries
- Diaphragmatic injuries
- Cardiac injuries.

**Pneumothorax**

Pneumothorax is the presence of air in the pleural cavity.

*Causes*

- **Spontaneous pneumothorax:** Occur due to rupture of emphysema blebs most commonly in smokers. Most common site is the apical region of lung.  
Treatment – needle aspiration for localized bleb rupture, thoracoscopy and pleurodesis is the definitive treatment option.
- **Iatrogenic pneumothorax:** Due to complication of surgical thoracic procedures such as lung or pleural biopsy, neck line insertion, positive pressure ventilation, needle thoracocentesis or liver abscess aspiration.

- **Traumatic pneumothorax:** Occurs as a result of blunt or penetrating thoracic trauma.

#### *Types*

- **Tension pneumothorax**

- *Pathophysiology*

- When the opening allows air to enter the pleural cavity during expiration but does not allow its drainage during inspiration, the pleural cavity gets filled with air which cannot escape [one way valve] and therefore increases intrapleural tension.
    - This results in positive intrapleural pressure which causes lung collapse first ipsilateral, then contralateral mediastinal shift and finally contralateral lung collapse if not treated.

- *Clinical features*

- Respiratory distress
    - Tracheal deviation to opposite side
    - Absent breath sounds on affected side
    - Distended neck veins and systemic hypotension
    - Subcutaneous emphysema on affected site.

- *Investigation*

This is a clinical diagnosis and **no investigation is necessary.**

- *Treatment*

- First take care of airway with cervical spine protection as per the ATLS protocol.
    - After ruling out airway obstruction or injury and the need of urgent intubation, take care of the breathing problem that the patient has (tension pneumothorax) according to the ATLS protocol
    - First step here is to do needle decompression in the midclavicular line in the second intercostal space to drain the air trapped in tension
    - This is followed by tube thoracostomy (intercostal tube drain) through midaxillary line in 5th or 6th intercostal space especially from a site separate from the wound site
    - The final step is wound closure. Always remember that the wound closure is done only after the two important steps of needle decompression and tube thoracostomy.

- **Open pneumothorax**

- *Pathophysiology*

- This is due to a large open wound which allows the pleural space to communicate to exterior and allow free entry as well as exit of air
    - Because it is two way wound, the risk of lung collapse and mediastinal shift is less urgent than in tension pneumothorax
    - This wound is also known as 'suckling chest wound'.

- *Clinical features*

- Respiratory distress
    - Tracheal deviation to opposite side
    - Absent breath sounds on affected side
    - Distended neck veins and systemic hypotension
    - Subcutaneous emphysema on affected site

– *Investigation*

This is a clinical diagnoses and **no investigation is necessary.**

– *Treatment*

- First take care of airway with cervical spine protection as per the ATLS protocol
- After ruling out airway obstruction or injury and the need of urgent intubation, take care of the breathing problem that the patient has (Tension pneumothorax) according to the ATLS protocol
- First step here is to occlusive dressing with **tape in three sides of the wound** and one side open to prevent it from forming tension pneumothorax
- This is followed by tube thoracostomy (Intercostal tube drain) through midaxillary line in 5th or 6th intercostals space especially from a site separate from the wound site
- The final step is definitive wound closure. Always remember that the wound closure is done only after the two important steps of three sided occlusive dressing and tube thoracostomy.

**Q4. Write a note on hemothorax.**

**Ans.** Hemothorax is accumulation of blood in the pleural cavity. It results most commonly from blunt or penetrating thoracic trauma.

Other causes include lung tumor, mesothelioma, pulmonary tuberculosis and rupture of hydatid cyst.

**Clinical features**

- Respiratory distress
- Tracheal deviation to opposite side
- Absent breath sounds on affected side
- Subcutaneous emphysema can be present on affected site.

**Investigation**

- Blood grouping and cross matching, hemoglobin determination
- Done only if the patient is hemodynamically stable. Otherwise directly proceed to thoracotomy
- Include chest X-ray, CECT chest.

**Treatment**

- First take care of airway with cervical spine protection as per the ATLS protocol
- After ruling out airway obstruction or injury and the need of urgent intubation, take care of the breathing problem according to the ATLS protocol.

**Hemodynamically stable patient**

- This is followed by tube thoracostomy (intercostal tube drain) through midaxillary line in 5th or 6th intercostals space especially from a site separate from the wound site
- Monitoring of output from the chest tube and deciding on thoracotomy or conservative management according to the patient condition.

**Indications of thoracotomy**

• **Massive hemothorax**

- Blunt chest trauma with >1500 mL blood on tube insertion or penetrating chest trauma with >1000 mL blood

- Or drainage of >33% blood volume on tube insertion
- Drainage of >200 mL/hour blood for 3 or more consecutive hours
- Caked hemothorax

- **Hemodynamically unstable patients.**

**Other indications:** Pericardial tamponade, cardiac herniation, massive air leak with inadequate ventilation or persistent lung collapse, esophageal injury, tracheobronchial injury, great vessel injury or aortic injuries.

- Further management depends on the cause that is identified during thoracotomy.
  - If the patient is stable after tube thoracocentesis, monitor the patient carefully for vitals monitoring, intake and output, especially urine output, hematocrit and hemoglobin determination
  - The chest tube is removed when the lung fully expands and the output is nil or nearly nil or < 50–100 mL serous for > 24 hours.

**Q5. Write a note on traumatic cardiac tamponade management.**

**Ans.** Cardiac tamponade is accumulation of blood in the pericardial space.

**Pathophysiology**

- The most common cause is penetrating injury to heart
- The blood starts accumulating in the pericardial space and because the pericardium is not distensible, the pressure in pericardial space increases to match that of the injured chamber
- Finally there is equalization of the pressure in all 4 chambers of the heart
- This leads to the clinical features of cardiac tamponade
- The acute event also compromises cardiac blood supply and can lead to subendocardial ischemia and arrhythmias.

**Clinical features [Beck's triad]**

- Hypotension
- Distended neck veins
- Muffled heart sounds
- Pulsus paradoxus
- Even 100 mL of blood if acutely collected can lead to death

**Management**

- First take care of airway with cervical spine protection as per the ATLS protocol
- After ruling out airway obstruction or injury and the need of urgent intubation, take care of the breathing problem according to the ATLS protocol.

**Further management depends on the clinical status of the patient and is as follows:**

- **Patient in cardiac arrest**

See if patient has indication for emergency department thoracotomy

- Salvageable postinjury witnessed cardiac arrest of < 5 minutes prehospital CPR in blunt trauma cases and < 15 minutes prehospital CPR in penetrating trauma cases
- Persistent postinjury hypotension (systolic < 90 mm Hg) with cardiac tamponade despite best possible resuscitation.

Otherwise manage the patient as per the outline of management of cardiac arrest as given in the question on cardiac arrest

- **Hemodynamically unstable patients should directly undergo emergency department thoracotomy**
- **Hemodynamically stable patients.**

#### **Investigation**

- Ultrasound through subxiphoid or parasternal window in the emergency department is confirmatory
- If diagnoses cannot be made on ultrasound, then the subxiphoid pericardial window procedure is done to evaluate and confirm the diagnoses. This is preferred as the incision can be extended to a median sternotomy if required.

#### **Treatment**

- Ultrasound guided pericardiocentesis through subxiphoid window is the life saving procedure followed by placement of pericardial catheter
- If the patient still has persistent hypotension after 2 liters crystalloid in adults and three fluid challenges of 20 mL/kg in children, then start blood transfusion and do emergency department thoracotomy.

#### **Important steps in emergency department thoracotomy**

- Left anterolateral thoracotomy in anterior 5th intercostals space starting from right end of sternum is done to carry out open cardiac massage and repair of cardiac tears
- Atrial cardiac tear is grasped in side biting satinsky clamp and repaired with continuous or interrupted monofilament sutures
- Ventricular tear is temporarily controlled using skin staplers or horizontal mattress sutures or foley's bulb occlusion
- The final repair is done in the operating room.

#### **Q6. Write a note on management of a patient of cardiac arrest.**

##### **Ans. Management of cardiac arrest is as follows:**

- Assess patency of airway and cervical spine stability
- Airway maintained using bag and mask ventilation or intubation and mechanical ventilation
- Once airway is taken care of look for carotid pulse.
- Start **cardiopulmonary resuscitation** if absent pulse:
  - If patient is intubated, set ventilator at 12 breaths/minute.
  - If not intubated, then give 2 breaths using bag and mask for every 30 chest compressions in 1 CPR cycle
  - 5 CPR cycles usually finish in 2 minutes
  - Chest compressions are given at 100/min with full chest recoil in between each compression
  - Chest compressions are given by heel of hand with all fingers and elbow extended fully and pressure applied through upper torso of the rescuer transmitted through shoulder, elbow and wrist. The fingers should not touch the chest wall. The heel of hand is kept on sternum at its lower aspect and compressions given.



- The chest compressions in pediatric age group are given using thumb or index and middle finger together.
- The displacement of chest wall should be around 4–5 cm vertically.
- While continuing this basic life support measure, attach ECG monitor, and automated defibrillator
- This defibrillator assesses the rhythm on its own and delivers the required shock. If it is not present then attach pain ECG monitor to look at rhythm and decide on further course of management
  - **If patient has asystole or pulseless electrical activity**, give 5 CPR cycles and then give epinephrine 1mg/kg or vasopressin 40 units and continue 2 minutes of CPR. Again assess rhythm and repeat the same for 2 more cycles. If any change, act accordingly. Consider atropine 1mg IV between cycles which can be repeated for 3 cycles
  - **If the patient has a shockable rhythm [ventricular fibrillation or tachycardia]**, then give shock and continue CPR if no change. Injections as above can be given in between shocks. AED decides the power of shock on its own. If it is manual defibrillator then use 200 joules biphasic current or 360 joules direct current.
  - **If the patient does not respond to second shock**, then consider antiarrhythmic drugs such as amiodarone 300 mg IV followed by CPR and if necessary, single repeat dose of 150 mg IV. Also, magnesium 1–2 g IV or lignocaine 1–1.5 mg/kg and if necessary 0.5–0.75 mg/kg can be given 2 or 3 more times to a maximum dose of 3 mg/kg.

**In postresuscitation phase**

- Avoid hyperthermia
- Keep a strict glycemic control
- Therapeutic hypothermia is of benefit in patients who remain comatose
- Avoid electrolyte imbalance
- Correction of cause.

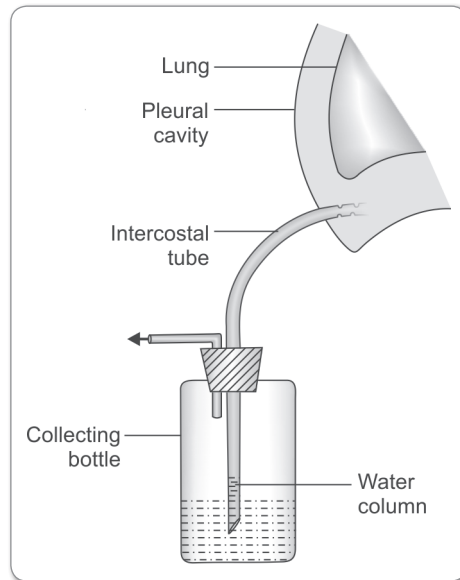
**Q7. Write a note on intercostal drainage using chest tube.****Ans. Indications of chest tube insertion**

- Pneumothorax
- Hemothorax
- Empyema
- After thoracotomy or thoracoscopy.

**Procedure**

- Informed consent is taken for the procedure
- If possible, patient is kept in 45° propped up position with head turned to opposite side and ipsilateral arm raised overhead
- Painting is done from opposite midclavicular line to the ipsilateral posterior axillary line and vertically from clavicle to 12th rib below and finally axilla and arm
- Then the local anesthesia is given in the 5th intercostal space just above the lower rib and incision is made of about 1–1.5 cm

- The incision is deepened upto muscles and then curved long artery forceps is used to separate the muscle layers
- The parietal pleura is then incised and chest tube inserted using a Kocher forceps
- The tube is then clamped and the closed end cut followed by connection of the tube to a water seal drainage bag prefilled with water upto the water mark
- If the drainage bag is not available, then the tube can be immersed in a saline bottle half filled with water
- The tube is fixed using a purse string suture. A suture that is not tied and is purse string type is also taken and simply draped around the tube to aid in wound closure when the tube is removed.



**Fig. 1:** ICD drainage

#### **Instructions to patient and relatives**

- To always keep the water seal drainage bag filled upto water mark
- Empty it only in presence of a supervisor nurse or doctor
- Look for movement of water column and inform if it is not moving
- Always keep the water seal drain bag below chest level
- Never keep it clamped as it can lead to tension pneumothorax
- Inform the doctor if patient experiences any respiratory symptoms.

#### **Removal of tube**

- Lung has expanded
- When the pus output is nil
- Blood output is nil
- Serous discharge is less than 100 mL/day for 2 consecutive days
- If it is post surgery tube, then it can be removed when the drain output is less than 300-350 mL/day because that is the normal pleural absorption capacity.

The tube is removed using the nontied purse string to close the wound and a tight occlusive dressing applied to prevent air leak either way.

### Complications

- Lung injury
- Bronchopleural fistula
- Bleeding from site
- Infection at insertion site, pleural space or lungs.

## PERIPHERAL ARTERIAL DISEASE FOR LOWER LIMB

**Q8. Describe the arterial anatomy of the lower limb.**

**Ans.**

**Arteries of lower limb are as follows:**

External iliac artery		Internal iliac artery
Deep circumflex artery Inferior epigastric artery (cremasteric A, pubic branch which is accessory obturator A) <b>Continues as femoral artery</b>		Obturator branch of anterior division supplies lower limb
<b>Branches of femoral artery</b>	<ul style="list-style-type: none"> <li>• Superficial circumflex iliac, superficial epigastric, Superficial external pudendal</li> <li>• Deep external pudendal, profunda femoris which gives 4 perforating arteries and medial and lateral circumflex femoral arteries</li> <li>• <b>Femoral then continues as popliteal artery</b></li> </ul>	<b>Anterior division of internal iliac artery gives</b> <ul style="list-style-type: none"> <li>• Superior vesical artery – remnant of the umbilical artery</li> <li>• Inferior vesical artery</li> <li>• Middle rectal artery</li> <li>• Internal pudendal artery – inferior rectal artery, deep and dorsal artery of penis, accessory pudendal artery in 10% cases.</li> <li>• Inferior gluteal</li> <li>• Uterine / defferential artery</li> <li>• Obturator artery</li> </ul>
<b>Branches of popliteal artery</b>	<ul style="list-style-type: none"> <li>• Anterior tibial artery – dorsalis pedis artery</li> <li>• Fibular artery – lateral calcaneal artery</li> <li>• 4 genicular arteries</li> <li>• Posterior tibial artery – medial calcaneal, medial and lateral planter arteries</li> </ul>	<b>Posterior division gives</b> <ul style="list-style-type: none"> <li>• Iliolumbar</li> <li>• Superior gluteal</li> <li>• Lateral sacral</li> <li>• Posterior branch</li> </ul>

**Q9. Enumerate the causes and risk factors of the peripheral vascular disease:**

**Ans.**

**Risk factors for peripheral arterial disease**

- Smoking in any form
  - Smoking index > 300
  - Pack year index > 40

- Diabetes
- Hypertension
- Dyslipidemia
- Hyperhomocysteinemia
- Hyperviscosity syndromes
- Male obese patients are also at increased risk
- Family history of vascular disease
- Personal history of myocardial infarction or stroke
- Hypercoaguable states (Factor V leiden mutation is the most common cause of congenital hypercoaguability and antiphospholipid antibody syndrome and smoking are the most common causes of acquired hypercoaguability)
- Postmenopausal status
- Prolonged physical inactivity.

#### **Common causes of peripheral arterial diseases**

- Atherosclerosis
- Burger disease
- Raynaud's disease
- Polyarteritis nodosa
- Takayasu arteritis
- Syphilitic arteritis.

#### **Difference between atherosclerosis and Buerger disease**

<b>Atherosclerosis</b>	<b>Buerger disease</b>
Age > 40 years	Age 20 – 30 years
Can occur without smoking	Smoking present
Venous involvement—thrombophlebitis, inflammation not seen	Venous involvement—thrombophlebitis seen
Only intima involved	Panarteritis, periarteritis, panphlebitis, perineuritis
Large and medium arteritis with extensive collaterals	Small arteries involved, no collaterals
Tree root appearance	Cork screw appearance
Distal run off present	Distal run off absent

**Q10. Discuss the clinical features and investigations in a patient with peripheral arterial disease (PAD).**

**Q11. Enumerate the clinical features of acute lower limb ischemia.**

**Ans.**

#### **Fontaine classification of peripheral arterial disease symptomatology**

- **Stage 1:** No clinical symptoms
- **Stage 2:** Intermittent claudication
  - Well compensated
  - Poorly compensated

- **Stage 3** : Rest pain
- **Stage 4** : Gangrene/ischemic ulcer.

### Rutherford classification of peripheral arterial disease symptomatology

- **0** – No symptoms
- **1** – Mild claudication
- **2** – Moderate claudication
- **3** – Severe claudication
- **4** – Rest pain
- **5** – Minor tissue loss
- **6** – Major tissue loss.

### Intermittent claudication

- Latin word claudio means “I limp”
- **Definition:**
  - Cramp like muscle pain
  - Due to accumulation of substance P due to inadequate blood flow
  - Not felt prior to first step and develop on exercise or walking
  - Decrease on rest
  - Can be reproduced by similar or less than previous exertion.
- **Boyd’s classification of intermittent claudication**
  - **Grade I** – patient continues to walk and the pain disappears
  - **Grade II** – pain continues, but patient also continues to walk
  - **Grade III** – Patient is compelled to take rest to relieve pain
  - **Grade IV** – rest pain.
- **Differences between vascular and neurogenic claudication**

Vascular claudication	Neurogenic claudication
<ul style="list-style-type: none"> <li>• Occurs after fixed amount of exercise</li> <li>• Not related to posture</li> <li>• Relief is rapid on rest</li> <li>• No relief on stooping</li> <li>• Sclerotomal distribution</li> <li>• Pallor/pulselessness/hypothermia can be associated</li> <li>• Occurs due to stenosis or occlusion of lower limb vessels</li> <li>• Straight leg raising test is negative</li> </ul>	<ul style="list-style-type: none"> <li>• Variable exercise produce pain</li> <li>• Prolonged maintained posture can produce pain</li> <li>• Relief is slow &gt; 30 minutes</li> <li>• Stooping can relieve pain</li> <li>• Dermatomal distribution of pain and sensory loss</li> <li>• Pallor, pulselessness, hypothermia is not associated</li> <li>• Occurs due to lumbar nerve root problem or cauda equina syndrome (spinal stenosis)</li> <li>• Straight leg raising test is often positive</li> </ul>

### Rest pain

- Ischemic neuritis
- Severe opioid resistant pain
- “Cry of dying nerves” due to ischemia of somatic nerves
- Seen on distal toes and metatarsal heads
- Increase on limb elevation and decrease on bringing leg to horizontal or dependent position.

**Acute ischemia**

- Duration of symptoms is less than 14 days
- Acute onset pain, paresthesia, rapidly progressive or established gangrene without the presence of chronic symptoms is acute ischemia
- Due to an embolic or thrombotic event.

**Chronic ischemia**

- Duration of symptoms is more than 14 days
- Pallor, paresthesia, purple hue of skin
- Pain, poikilothermia
- Ulceration in digits
- Wasting of muscles, dry skin, loss of hair, loss of subcutaneous fat

**Critical limb ischemia [CLI]**

Any of following suggest CLI

- Fontaine stage 3 and 4 (rest pain, ischemic ulcers and gangrene)
- Buerger angle  $< 20^\circ$
- Capillary refill time  $> 30$  seconds
- Systolic ankle pressure  $< 50$  mm Hg or systolic toe pressure  $< 30$  mm Hg
- Ankle brachial pressure index  $< 0.5$
- Delayed reactive hyperemia time.

**Pregangrene**

Criteria include one or more of following

- Rest pain
- Pallor on limb elevation
- Congestion in dependent position
- Scaling of skin and guttering of veins
- Coldness, tenderness and hyperesthesia.

**Investigations in a case of PAD****Diagnoses***Clinical findings*

- History of above symptoms
- History to rule out the risk factors for peripheral arterial disease
- Inspection reveals signs of ischemia as mentioned above
- Palpation is mainly to look for the level of absent or diminished pulses and examination of the gangrene or ulcer that is present
- If an ulcer is present, then see if it is arterial or venous ulcer or ulcer due to some other cause.

**Ulcer**

Breach in the continuity of skin and/or mucous membrane due to molecular death of tissue.

Venous ulcer	Arterial ulcer
Occur in gaiter area	Occur on toes, heel, dorsum of feet
More common in women	More common in man
Thrombophilia, positive family history predispose	Smoking, diabetes, hyperlipidemia, hypertension predispose
History of varicose veins, DVT	History of claudication, rest pain
Edema, pigmentation, deformity, ankylosis, lipodermatosclerosis can be seen	Signs of ischemia can be present
Ulcer is usually superficial, painful initially but painless chronically	Ulcer is deep and severely painful
It has irregular margin and has pink granulation tissue and minimal slough	It has regular margins and necrotic slough with no granulation
Limb elevation and exercise relieves pain	Limb elevation and exercise aggravates pain

- **Tests for lower limb ischemia** include Buerger's test, capillary filling and refilling, Reactive hyperemia time, Fuschig crossed leg test
- Sensory and motor examination is to be done and examination of lymph nodes should not be forgotten
- Power of muscles is to be checked and grading is as follows:

0	Paralysis
1	Flicker
2	Movement with gravity eliminated
3	Movement against gravity
4 –	Movement against slight resistance
4 0	Movement against moderate resistance
4 +	Movement against strong resistance
5	Movement against full resistance
Not testable	----

- **ABPI (ankle brachial pressure index)** = Systolic pressure at the ankle of the leg/systolic pressure at arm (greater of 2 arms)

#### Grades of ABPI

> or = 1.4	Diabetes/end stage renal disease
1 – 1.3	Normal
< 0.9 – 0.71	Risk of MI increases
< 0.7 – 0.51	Claudication
< 0.5 – 0.31 [CLI]	Rest pain
< 0.3 [CLI]	Gangrene

- **TBPI (Toe brachial pressure index)** = Toe systolic pressure using Doppler/systolic pressure at arm (greater of 2 arms)
  - Normal – 0.8 – 0.9

- Claudication – 0.2 – 0.5
- Critical limb – < 0.2
- **Stress exercise testing**
  - Positive when on exercise,**
    - Fall in ABI > 0.2
    - Fall in ankle systolic pressure > 20 mm Hg
    - Failure of ABI to normalize in 3 minutes
    - Disappearing pulse (Pulse present normally but disappears after exercise to claudication and returns when the patient rests).

### Investigations

- **Doppler** is the first and most important investigation in peripheral arterial disease and is discussed in the radiology section in detail.

**Remember though** Doppler alone is just sound signals. Duplex is what we see in radiology rooms on the screen of ultrasound machine. Duplex is nothing else but Doppler with B mode ultrasound.

*Other investigations include*

- **Transcutaneous pulse oximetry** at toes
- **Angiography**
  - Invasive angiography is the gold standard investigation
  - Opposite common femoral artery or left brachial artery access is used
  - Total contrast required is around 75 – 100 mL
  - Caution exercised to stop metformin, ACE inhibitors or diuretics if patient is on them for upto 48 hours after the procedure
  - It is done in following important sites:
    - Abdominal aorta and origins of celiac A, superior and inferior mesenteric A
    - Aortic bifurcation, common iliacs, external and internal iliac arteries
    - Opposite lower limb arteries and finally ipsilateral lower limb arteries
  - The options more commonly used now for avoiding an invasive procedure are:
    - **CT/MR angiography**
    - **CO<sub>2</sub> angiography:** Used CO<sub>2</sub> instead of contrast used especially in the patients with renal insufficiency
    - **Intravascular ultrasound with/without Doppler.**

**Q12. What is gangrene? Discuss its causes.**

**Write a note on types of gangrene.**

**Ans.**

### Gangrene

- Death of macroscopic portion of tissue with or without superadded putrefaction
- It is black because of breakdown of hemoglobin and formation of iron sulfide within the tissue
- The line of demarcation is area of hyperesthesia and hyperemia



**Causes of gangrene**

- Peripheral arterial disease (all causes as enumerated above)
- Deep vein thrombosis
- Diabetic gangrene
- Vascular injury and traumatic gangrene
- Neurological diseases (paraplegia, hemiplegia, leprosy, syringomyelia, syphilitic neuritis)
- Frost bite, electrical or thermal burns, chemical injury and gangrene
- Infections (gas gangrene, moist gangrene, Fournier gangrene, Meleney's gangrene)

**Dry gangrene**

- Dessicated, mummified, shrivelled tissue due to gradual slowing of the blood stream
- It is noninfected gangrene.
- There is a clear-cut line of demarcation formed by hyperesthetic, healthy granulation tissue between living and dead.
- Process is called aseptic ulceration.

**Wet gangrene**

- Both arterial and venous supply is blocked with superadded infection and putrefaction
- It is soft and boggy and is infected
- There is congestion, edema, pus discharge and no clearcut line of demarcation
- Process is called septic ulceration
- **When wet gangrene occurs due to gas forming organisms, it causes gas gangrene.**

**Diabetic gangrene**

- Occurs because of the combination of three factors—ischemia due to atheroma, peripheral neuropathy and immunosuppression due to the diabetes.
- It can cause dry, wet or gas gangrene.

**Q13. Discuss the management of a case of peripheral arterial disease.****Outline the management of a patient with acute limb ischemia.****Write the management of a patient with critical limb ischemia.****Ans. Management**

Management of a case of peripheral arterial disease for lower limb is a difficult topic to address especially for the undergraduates. What is done here is give you all the outline for managing the different entities associated with peripheral arterial disease and then discuss few of them in detail.

**1. Intermittent claudication management****a. No significant disability with only mild symptoms**

Monitor annually or 6 monthly for lower limb/ central nervous system and cardiovascular system arterial disease and do risk factor modification.

*Risk factor modification*

- Smoking cessation
- Hypertension and diabetic control

- Weight reduction
- Control of hyperlipidemia
- Care of feet (chiropody)
- Exercises to claudication point

**b. Claudication with lifestyle limiting symptoms but no inflow disease (aortoiliac disease) on investigation**

- Risk factor modification as above
- Exercise to claudication point in a supervised manner
- Drugs – pentoxifylline, cilostazole.

*Cilostazole*

- Inhibits platelet aggregation, inhibits smooth muscle proliferation, is a phosphodiesterase type 3 inhibitor, inhibits HDL and triglyceride synthesis and decreases their levels, and increases vasodilatation
- It is better than pentoxifylline and improvement ranges from 35–100%.
- It is given as 100 mg BD orally and to be given for at least 3 months before response evaluation.
- It is contraindicated in congestive heart failure.

*Pentoxifylline*

- It is a phosphodiesterase inhibitor as well as inhibitor of adenosine 2 receptors
- Improves red blood cell deformability (rheologic effect)
- Reduces blood viscosity and
- Decreases the potential for platelet aggregation and thrombus formation.

*Other drugs*

- **Statins** when LDL > 130 mg/dL, HDL < 40 mg/dL. Triglycerides > 150 mg/dL
- **Antihypertensive drugs** when BP is > 130/85 mm Hg
- **Anti hyperglycemics** when HbA<sub>1c</sub> > 7
- **Aspirin + clopidogrel** for prevention of myocardial infarction, stroke etc.
- **Analgesics**
- **Beta blockers** as cardioprotective agents are also used sometimes.

**c. Claudication with lifestyle limiting symptoms and inflow disease (aortoiliac disease).**

Further imaging to identify the disease and its level and do endovascular/surgical management.

**In all the types**, if the condition improves manage as in a) and if the condition deteriorates, Investigate b) as c) and reinvestigate and manage patients accordingly with c).

**2. Management of a case of acute limb ischemia**

In all the patients, first management is **starting systemic heparin** and then manage as follows:

a.	Viable limb Not threatened immediately	Investigate and plan for management as revascularisation
b.	Threatened limb but salvageable	

Contd...

Contd...

<b>c.</b>	Limb only salvageable by emergency surgery	No time for imaging Attempt revascularization
<b>d.</b>	Non salvageable limb (gangrene)	Amputation

*Management options*

Endovascular therapy	Thrombolysis with/out mechanical thrombectomy	5 mg TPA bolus
	Thrombectomy	
Surgery	Thrombectomy or	With/out endarterectomy
	Embolectomy	
Both types of therapy are to be preceded by systemic anticoagulation as mentioned earlier		

**3. Management of a case of confirmed critical limb ischemia**

<b>Salvageable</b>		Revascularization as appropriate
<b>Not salvageable</b>	Patient has extreme pain and is surgically fit	Amputation
	Patient is not surgically fit or Patient has a painless stable lesion	Medical management

**4. Management of a case of chronic limb ischemia**

Aim is revascularization. Amputation is reserved for established gangrene and failure of revascularization treatment.

**Options for revascularization** in a case of chronic limb ischemia are as follows:

<b>Endovascular treatment</b>	Atherectomy
	Angioplasty alone
	Angioplasty and stenting
<b>Surgery</b>	Thromboendarterectomy
	Anatomic bypass (e.g. Femoropopliteal)
	Extra-anatomic bypass (e.g. Axillofemoral)

Always remember that as long as the endovascular treatment does not negatively affect the patient's surgical management if required in future in case of restenosis or reocclusion, endovascular management is always to be the first option in all cases of peripheral arterial disease.

**Q14. Write a note on Buerger disease.**

**Discuss the clinical features and management of a patient with thromboangiitis obliterans.**

Ans.

**Buerger disease**

- Buerger disease is progressive segmental inflammation of small and medium sized arteries with periarteritis, panarteritis, periphlebitis, thrombophlebitis and panneuritis

- It is seen in male smokers between 20 and 40 years old and more common in Jews
- Etiology is same as for other peripheral arterial diseases with smoking being the most important risk factor. Also autoimmune etiologies have been suggested but only smoking is the most established risk factor. Rickettsial infection and vasculitis have also been suggested etiologies
- The clinical features, diagnoses and management is as for the above symptoms and signs of peripheral vascular disease

(**Always remember:** Amputation, sympathectomy and omental transfer operations are always asked in PVD section and these are the grey areas. So they are now addressed in following few questions for clarity.)

Endovascular surgery is an upcoming topic and is discussed in brief too.)

### **Q15. Write a note on indications and complications of amputation.**

**What is amputation? Write the criteria for an ideal amputation stump.**

**Ans.** Amputation is an operation done to remove the limb or part of limb through one or more bones. When done through joints, it is called disarticulation.

#### **Indications**

##### *Congenital anomalies*

- Supernumerary digits
- Arteriovenous fistula.

##### *Inflammatory*

- Osteomyelitis
- Madura foot
- Refractory septic arthritis.

##### *Traumatic*

- Most common indication
- Traumatic upper limb machinery injuries and accidents account for most cases.

##### *Neoplasm*

- Soft tissue sarcoma with bone invasion
- Osteosarcoma/ fibrosarcoma/ chondrosarcoma
- Neuroma.

##### *Vascular cause*

- Acute severe ischemia with no revascularization options
- Chronic irreversible ischemia
- Refractory cases of frost bite
- Failure of revascularization procedures
- Dry gangrene/wet gangrene/gas gangrene
- Diabetic foot with gangrene or irreversible foot sepsis.

#### **Types of amputation**

- Guillotine type or flap type—circular, elliptical, tennis racquet incision flap techniques are available

- End bearing or side bearing
- Weight bearing or non-weight bearing.

**Criteria of ideal stump**

- The suture line should not fall in the line of pressure points or prosthesis fitting
- It should not be too redundant or floppy
- It should not be closed under tension
- It should be optimum length to allow mobility and prosthesis fitting
  - **Arm** – 20 cm from acromial tip
  - **Forearm** – 20 cm from tip of olecranon
  - **Thigh** – 25 cm from tip of greater trochanter
  - **Leg** – 15 cm from tibial tuberosity
- There should be no bony spur projecting through the stitch line or flap
- The stump should be well rounded and smooth
- The scar should be mobile and nonadherent to underlying tissues
- The level of amputation should be decided based on the indication and function of the stump after the procedure.

**Complications of amputation****Early**

- Bleeding – can be primary, reactionary or secondary
- Recurrence of gangrene or infection
- Wound infection
- Flap necrosis
- Deep vein thrombosis and pulmonary embolism.

**Late**

- Painless phantom limb
- **Pain**
  - **Painful phantom limb**
    - Patient feels the pain/tingling/warmth/cold sensation in the limb that he/she does not now possess
    - It is more common in women and more common with upper limb amputations
    - The cause is not clear but is thought to be due to lack of feedback to central impulses towards the affected limb or lack of sensory input of the affected limb from spinal centers or a peripheral effect due to damaged nerves in the stump
    - Relief of pain prior to the surgery reduces the chances of phantom limb postoperatively
    - Treatment is difficult but includes medicines such as gabapentine, carbamazepine, amitriptyline, procedures such as transcutaneous electrical nerve stimulation (TENS), mental imagination techniques such as mirror therapy or imaginery use of the amputated limb, massage or acupuncture.
  - **Stump neuroma** – proliferation of cut nerve ending
  - Painful stump scar

- **Causalgia** – Persistent pain at the stump end caused by the sensations through the damaged nerves
- **Jactitation pain** – Pain due to spasm of muscles at the stump end
- Flap necrosis
- Pressure necrosis of stump and ulceration
- Psychiatric problems: Depression, anxiety, suicidal tendency.

#### Different amputations to remember

Upper limb	Lower limb
<b>Digit amputation</b> <b>Forearm amputation</b> <b>[Krukenberg amputation]</b> – Claw formation with radius and ulna. No need of prosthesis <b>Below elbow amputation</b> <b>Above elbow amputation</b> <b>Forequarter amputation</b> – anterior approach [Beger] or posterior approach [Littlewood]- in both approaches, scapula and lateral third of clavicle are to be removed	<b>Ray's</b> – Through metatarsal head <b>Gillies</b> – Transmetatarsal <b>Lisfranc</b> – Tarsometatarsal <b>Chopart</b> – Midtarsal with talonavicular and calcaneocuboid disarticulation <b>Syme's</b> – Through tibia and fibula <b>Modified Syme's</b> – Elliptical incision with heel flap supplied by medial and lateral calcaneal arteries <b>Pirogoff</b> – Posterior calcaneum preserved, rest is like Syme's <b>Burgess</b> – Below knee with minimum length 8 cm from tibial tuberosity for prosthesis. Long posterior flap <b>Peg-leg</b> – Below knee amputation with long anterior flap <b>Gritti-Stokes</b> – Trans femoral condylar amputation <b>Above knee</b> – Minimum femur length 10 cm. Both flaps are of equal length <b>Hindquarter amputation (hemipelvectomy)</b> – approach can be anterior (Tennis racquet or Boyd) or posterior (Solcum). It can be <ul style="list-style-type: none"> <li>• <b>Standard</b> – Entire innominate bone removed</li> <li>• <b>Extended</b> – Sacrum also removed</li> <li>• <b>Internal</b> – Limb preserved but the innominate bone removed</li> <li>• <b>Conservative</b> – Ilium and pubis preserved</li> </ul>

**Q16. Write in brief about the role of sympathectomy in modern surgical practise.**

**Discuss the role of sympathectomy and omental transfer operations in peripheral vascular disease management.**

**Ans.**

#### Sympathectomy

##### Indications

- Peripheral arterial disease (atherosclerosis/Buerger) – Atherosclerosis with rest pain and no revascularization option. In these cases, sympathectomy can relieve pain, increase the collateral blood flow to superficial tissue and skin and if amputation is required, it limits the extent of amputation
- Causalgia
- Hyperhydrosis
- Vasospastic hand disorders such as Raynaud disease – T3, 4 sympathectomy
- Intractable facial blushing/sweating – T2,3 sympathectomy

- Erythrocyanosis
- Acrocyanosis.

#### *Mechanism*

It mainly acts to increase the blood supply to skin and superficial tissue only. It does not have effect on deep circulation system and muscle blood supply.

#### *Sites*

- Thoracodorsal sympathectomy = Cervical sympathectomy = Cervicothoracic sympathectomy
- Lumbar sympathectomy.

#### **Thoracodorsal sympathectomy**

- Open approach—Anterior/axillary/supraclavicular/posterior
- VATS (video assisted thoroscopic surgery)
- Complications—Injury to stellate ganglion and Horner syndrome, compensatory truncal hyperhidrosis can occur. Therefore care should be taken to prevent C8-T1 ganglion injury.

#### **Lumbar sympathectomy**

- Unilateral—L1-3/4
- Bilateral—L1 of one side needs to be preserved to prevent retrograde ejaculation
- Approach can be open or laparoscopic and in both approaches, the operation can be extraperitoneal or intraperitoneal approach. The intraperitoneal approach is specially used in cases for bilateral cases
- Complications—Paralytic ileus, vessel or bowel injury, Injury to ureter or genitofemoral nerve.

#### **Omental transfer and omentopexy**

- **Mainly indicated in patients with Buerger's disease** with no revascularization option and incapacitating pain with failure of lumbar sympathectomy also
- The omentum is taken as a pedicled flap to the affected limb by tunnelling through the inguinal region and this lengthening of omentum is based on the gastroepiploic arteries
- The transferred omentum increases the local collateral circulation and promotes neovascularization
- This return of blood supply is believed to relieve the symptoms.

#### **Q17. Write a note on endovascular management of peripheral arterial disease.**

##### **Ans. Endovascular surgery**

#### *Equipment [In sequence of use]*

- **Needles** – 18 or 21 gauge
- **Guidewire** – has stiff inner core, coated surface and straight/ angled or curved tip
- **Hemostatic sheath** is to protect the vessel wall from injury and is with a side port for injecting contrast or heparin
- **Catheter** – Atherectomy – excisional (rotating cutters) or ablational (laser using ultraviolet xenon energy), angiography, balloon dilatation with/ out stenting
- **Balloon** – Low compliance balloon is more rigid and offer greater dilating force

- Can be cutting balloon (balloon with 3–4 atherotomes mounted on its surface) or cryoplasty balloon (balloon which is inflated with nitrous oxide)
- **Stents.**

#### Indications

- Inadequate angioplasty (> 30° residual stenosis)
- After subintimal atherectomy/angioplasty
- Complication of angioplasty – Intimal flap/arterial dissection/restenosis

*Material* – Stainless steel, tantalum, cobalt based alloy, nitinol (nickel and titanium alloy)

#### Types

- **Self expandable**—Nitinol stents deployed by removing a restraining sheath, are long and used for long, tortuous lesions and vessels.
- **Balloon expanded**—Nitinol or stainless steel deployed and expanded using balloon, mainly used to treat short segment ostial calcified lesions.
- **Drug eluting stents**—Nitinol with drug coating – Sirolimus/paclitaxel

### Q18. Enumerate the types of vascular grafts.

Ans.

<b>Bioprosthetic</b>	Autograft	<ul style="list-style-type: none"> <li>• Saphenous vein</li> <li>• Left or right internal mammary artery</li> <li>• Hypogastric artery</li> <li>• Radial artery</li> <li>• Right gastroepiploic artery</li> </ul>
	Homograft	<ul style="list-style-type: none"> <li>• Human cadaver</li> </ul>
	Xenograft	<ul style="list-style-type: none"> <li>• Porcine</li> <li>• Bovine</li> </ul>
	Tissue engineering	
<b>Synthetic</b>	Stent grafts	<ul style="list-style-type: none"> <li>• Nitinol with ePTFE or polyester fabric covering used for thoracic or infrarenal aortic aneurysm repair/traumatic arterial disruption or AV fistulas repairs</li> </ul>
	Nontextile	<ul style="list-style-type: none"> <li>• Expanded PTFE (ePTFE)</li> <li>• Polyurethane</li> </ul>
	Textile	<ul style="list-style-type: none"> <li>• Woven dacron – less stiff, less stable</li> <li>• Knitted dacron – more porous, more stable, more easy to handle</li> </ul>

### Q19. What is diabetic foot? Discuss its clinical features and management.

Ans.

#### WHO definition

“Any infection, ulceration and/or destruction of deep tissue that comes along with neurologic abnormalities and/ or with/ out different stages of arterial closure disease in the lower limb”.

**Incidence** – 12 – 25 % lifetime risk of ulcer.



**Pathophysiology**

<b>Neuropathy</b>	<ul style="list-style-type: none"> <li>• Sensory—loss of protective sensation</li> <li>• Motor—muscle atrophy, foot deformity, altered biomechanics</li> <li>• Autonomic—loss of sweating, cracks and fissures, alteration of neurologic regulation of blood supply</li> </ul>
<b>Vasculopathy</b>	<ul style="list-style-type: none"> <li>• Microangiopathy</li> <li>• Accelerates atherosclerosis and atheroma formation</li> </ul>
<b>Infections</b>	<ul style="list-style-type: none"> <li>• Immunosuppression</li> </ul>

**Kobe university classification for Asian population based on pathophysiology**

<b>I</b>	Mainly peripheral neuropathy	Pressure relief
<b>II</b>	Mainly peripheral arterial disease	Revascularization
<b>III</b>	Mainly infection	Early debridement
<b>IV</b>	I + II + III	Case to case management including above three measures

**Classifications of diabetic foot***1. Maggitt's classification*

0	Foot symptoms, only pain
1	Superficial ulcer
2	Deep ulcer, bone not involved
3	Ulcer with bone involvement
4	Forefoot gangrene
5	Full foot gangrene

*2. Wagner classification*

0	No open foot lesion
1	Presence of partial or full thickness superficial ulcer
2	Ulcer extends to deep fascia, ligaments, tendons, or joint capsule without osteomyelitis or abscess
3	Presence of deep ulcer with abscess, osteomyelitis or joint sepsis
4	Gangrene localized to forefoot or heel
5	Extensive gangrene

*3. Modified Wagner or Depth – ischemia classification*

Depth		Ischemia	
0	The at-risk foot: Previous ulcer, neuropathy, deformity that predispose to ulcer	A	Not ischemic
1	Superficial ulceration, not infected	B	Ischemia present but no gangrene
2	Deep ulceration exposing tendon or joint with/ out superficial infection	C	Partial / forefoot gangrene
3	Extensive ulceration with exposed tendon/ joint and/or infection	D	Complete gangrene

**Example:** Patient with ischemic gangrene and noninfected superficial ulcer will be labelled as “1B”.

*4. University of Texas San Antonio classification*

	0	1	2	3
A	Pre or post ulcer epithelialized lesion	Wound not involving tendon, capsule or bone	Only bone not involved	Bone involvement present
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	B + C	B + C	B + C	B + C

**Example:** Patient with ischemic gangrene and noninfected superficial ulcer will be labelled as “1C”.

**Admission criteria**

*All limb threatening infections:*

- Edematous foot with full thickness ulcer that probes to bone or communicates with foot compartments with gangrene/purulence
- 2 cm of surrounding cellulitis or lymphangitis
- Systemic signs of toxicity (fever, leucocytosis, tachycardia, hyperglycemia, metabolic instability).

**Management of a patient with diabetic foot**

*Investigations*

- **Diabetes workup**—Premeal sugar level charting, HbA<sub>1c</sub> levels, 24 hour urine sugar and protein, ultrasound abdomen to look for diabetic nephropathy, fundus examination for diabetic retinopathy.
- **Infection screening**—Wound culture, blood culture, total and differential leukocyte count, ESR, C-Reactive protein levels, urine culture
- **Blood investigations**—Hemoglobin levels, electrolyte levels, kidney and liver function tests, platelet counts
- **Biochemistry**—Lipid profile, urine routine and microscopic analysis
- **Radiography**—X-rays of the affected limb, MRI to rule out osteomyelitis if suspected, bone scintigraphy (technetium 99 MDP, gallium, sulfur colloid scans can all be used)
- **Pathology**—Deep tissue biopsy and cultures, Bone biopsy to confirm osteomyelitis.

**Treatment**

- Management of diabetes, smoking, hypertension, hyperlipidemia and obesity
- Antibiotic therapy is started for all patients with limb threatening infections as well as all patients with infection or osteomyelitis
- Wound debridement and lavage with saline followed by hemostasis and wet to dry dressing
- The affected foot compartments should be opened generously using appropriate incisions to prevent compartment syndrome in foot and leg
- Well padded dressing from knees to toes
- Maintain foot hygiene

- Polymethylmethacrylate beads with antibiotic loaded cement (PMMA – ALC) can also be used to improve healing. The beads are coated with gentamicin/tobramycin or gentamicin and filled into the gaps in wound after debridement and then well padded dressing applied
- The management of diabetic ulcer, gangrene, peripheral arterial disease is as discussed above
- The role of amputation is also discussed above
- This is in short the management of a patient of diabetic foot.

## PERIPHERAL VENOUS DISEASES

**Q20. Write the anatomy of the venous system of lower limb.**

**Ans.**

### **Venous drainage of lower limb**

Comprises of superficial system, deep system and a perforator system which is the communication between the two.

### **Superficial system**

- Dorsal venous arch medially forms the great saphenous vein and laterally the short saphenous vein
- The great saphenous vein passes anterior to the medial malleolus, is accompanied by saphenous nerve, passes medial to knee, opens after piercing the cribriform fascia into the common femoral vein at the saphenofemoral junction
- The short saphenous vein passes posterior to lateral malleolus, is accompanied by sural nerve and terminates into the popliteal vein.

### **Deep system**

- Deep plantar venous arch forms the medial and lateral plantar veins which terminate into paired posterior tibial veins. The dorsalis pedis veins forms the paired anterior tibial veins
- The soleal sinuses also terminate into the anterior and posterior tibial veins
- These paired anterior and posterior tibial veins end into the popliteal veins. The popliteal vein also receives tributaries from gastrocnemius veins and short saphenous vein
- It then passes through the adductor magnus window, from where it is termed as superficial femoral vein
- Superficial femoral vein receives the profunda femoris vein and becomes the common femoral vein which receives great saphenous vein and ends as external iliac vein finally to common iliac vein and inferior vena cava.

### **Perforator system**

Upto 100 perforators have been identified.

- **Calf**
  - 5, 10, 15 cm from medial malleolus
  - 5, 12, 17 cm from lateral malleolus

- **Foot perforators** and
- **Mid thigh perforators**

Other named perforators

- **Cockett I,II,III perforators** – connect posterior arch vein to posterior tibial vein
- **Boyd perforator** – connect great saphenous vein to gastrocnemius vein
- **Dodd perforator** – connect great saphenous and superficial femoral vein
- **Hunterian perforator** – connect great saphenous and superficial femoral vein

**Q21. Enumerate the risk factors for varicose veins and discuss its clinical features.**

**Ans.**

**Risk factors for varicose veins**

- Prolonged standing
- Obesity
- Deep vein thrombosis
- Other causes are as follows:

Primary varicose veins	Secondary varicose veins	Congenital deformities
<ul style="list-style-type: none"> <li>• Defective valves</li> <li>• Can be due to thrombosis or inflammation of the veins</li> </ul>	<ul style="list-style-type: none"> <li>• Pregnancy</li> <li>• Deep vein thrombosis</li> <li>• Pelvic masses (ovarian/uterine/cervix/rectum)</li> <li>• Retroperitoneal masses or retroperitoneal fibroses</li> <li>• Arteriovenous fistulas</li> <li>• Family history, female sex</li> </ul>	<ul style="list-style-type: none"> <li>• Congenital Arteriovenous malformations</li> </ul>

**Clinical features**

*Symptoms*

- Visible veins in legs or thighs
- **Venous claudication** – aching sensation in legs and heaviness that increases when the legs are kept in dependent position for prolonged periods and is relieved on elevation of limb and exercise. Bursting pain indicates the presence of deep vein thrombosis
- Venous ulcer in the gaiter area. Features are described in the table above
- Gangrene
- Pruritus, pigmentation
- Edema of lower limb.

*Signs and tests*

- All above findings are confirmed
- **Tests**

Saphenofemoral incompetence	<ul style="list-style-type: none"> <li>• Trendelenburg 1 test</li> <li>• Schartz tap test</li> <li>• Morrissey cough impulse test</li> </ul>
Perforator incompetence	<ul style="list-style-type: none"> <li>• Trendelenburg 2 test</li> <li>• Pratt test</li> <li>• Fegan test</li> <li>• Multiple tourniquet test</li> </ul>

Deep veins	<ul style="list-style-type: none"> <li>• Perthe test</li> <li>• Modified perthe test</li> </ul>
Deep vein thrombosis	<ul style="list-style-type: none"> <li>• Homan's test</li> <li>• Moses test</li> <li>• Pratt test</li> </ul>

**Other important features**

- Ankle flare
- Ulcer in gaiter area
- Saphena varix at SFJ

**Classification of varicose veins and associated diseases**

**CEAP** [Clinical, Etiological, Anatomical, Pathophysiological classification]

**Q22. Write a note on management of a patient with varicose veins.****Discuss the treatment options for a patient with varicose veins.****Ans. Investigations**

- **Venous ultrasound** – more informative than venous Doppler because compressibility is seen in venous ultrasound  
Important steps in evaluation include:
  - Evaluation of perforator system and deep venous system in supine position and
  - Evaluation of varicosities and perforator incompetence in standing position
  - 4 mm size of perforators and reflux in perforators is called incompetence in venous ultrasound evaluation
- **Venography** is the gold standard evaluation technique but seldom used nowadays.
- Most patients are managed using ultrasound or Duplex evaluation only.

**Complications of varicose veins**

- Ulceration
- Eczema
- Pigmentation
- Calcification and calciphylaxis
- Lower limb edema
- Lipodermatosclerosis
- Champagne bottle leg
- Venous gangrene
- Bleeding from rupture of varicosities
- Superficial thrombophlebitis.

**Treatment modalities for varicose veins****1. Radiofrequency ablation (VNUS closure)**

- **Principle:**
  - High frequency, long wavelength, alternating electric current (460–480 kHz)
  - Electrode itself does not supply heat. The ionic agitation creates frictional heat which increases the surrounding tissue temperature to 50 – 100°C that produces coagulative necrosis

- It also produces conduction heat that causes slower damage of the tissues remote from the electrode tip
- Temperature greater than 100–110°C cause tissue carbonisation and vaporization with loss of ions thus stopping the current and acting as insulation
- These reactions in vein cause denaturation of vein wall collagen – leads to inflammation—fibroses and finally occlusion of vein.
- **System :**
  - **Generator:** Monopolar or bipolar system. Bipolar has the advantage of being safe, avoids complications due to earthing electrode and metallic implants are not a contraindication. Also it allows the use of higher current densities
  - **Electrodes:** Umbrella type/Christmas tree type, wet tip/cool tip type – longer duration of current flow and larger area of ablation
  - **Technique:** Percutaneous (USG/CT/MRI guided), open or laparoscopic. Used with USG guidance while treating varicose veins
  - **Tumescent local anesthesia** is used
    - Ringer lactate or normal saline is combined with lignocaine, epinephrine and bicarbonate to produce 0.1% solution
    - Acts as anesthetic, heat sink (insulator), vein lumen is compressed by it, thus increase energy transfer and decrease power requirements.
- **Indication**  
**Can be used for great saphenous vein and short saphenous vein.** The side branches that remain can be managed with phlebectomy/foam sclerotherapy
- **Benefits:**
  - Less pain, bruising and scarring
  - Early return to routine work
  - OPD procedure < 2 minutes and performed through a tiny incision in lower leg
  - 97.4% efficacy
- **Complications:**
  - **Early:** Pain, failure to ablate, thrombophlebitis, heat induced thrombosis, DVT (avoided by keeping the tip of the ablation catheter at least 2 cm away from the saphenofemoral junction)
  - **Late:** Skin pigmentation, recurrence, nerve damage and paresthesia.

## 2. Endovenous laser therapy (EVLT)

- 810 nm diode laser used with 700°C laser energy
- Uses tumescent local anesthesia
- Procedure is completed in 2 hours in OPD setting
- **Indication:** Approved for great saphenous vein. But cannot be used for deep veins from below knee to groin
- Again the side branches are managed with YAG laser (1064 nm), foam sclerotherapy or phlebectomy
- **Benefits:** Less pain, less bruising, low incidence of saphenous neuritis
- **Complications:** DVT, infection, bleeding, scarring, recurrence, burns.

### 3. Treatment of superficial small varicosities

- YAG laser (1064 nm)
- Foam sclerotherapy
- Microphlebectomy
- Transilluminated phlebectomy (TriVex system).

### 4. Spider vein treatment

- **USG guided foam sclerotherapy:** Very fine needle is used to inject a medication directly into spider vein or < 3 mm varicosities
- Can be used for treating spider veins, varicosities, saphenous trunks and venous malformations
- Agents: Sodium tetradecyl sulfate/polidochanol/hypertonic saline either of the three with air to create foam using Tessari method. The bubble size less than 250 micrometer is highly interactive and is preferred
- Mechanism: Endothelial damage, vascular sclerosis, rapid thrombus formation
- Total volume per session < 10 mL and single site injectate < 0.1 mL
- Complications: Transient visual disturbance, tightness in chest or discomfort.

**New modality:** Unipolar thermocoagulation for spider veins and telangiectasia.

### 5. Surgery

GSV varicosities with SFJ incompetence	SFJ ligation and stripping upto knee [Trendelenburg operation]
SSV varicosities with SPJ incompetence	SPJ ligation with/out stripping
Incompetent perforators	Perforator ligation [Subfascial "Cockett and Dodd" and suprafascial "Linton" or Subfascial endoscopic perforator surgery {SEPS}]
Bunch varicosities	Stab phlebectomy
Deep veins	Direct venous reconstruction [femorofemoral bypass (Eduardo Palma) or saphenofemoral bypass] or Kistner procedures (direct valvuloplasty or venous segment transfer)

### Management outline

#### *Asymptomatic or inoperable cases*

Risk factor reduction and lifestyle changes as follows:

- Avoidance of prolonged standing
- Limb elevation during rest and at night
- Calf muscle strengthening exercise like bicycling in air and walking training
- Elastic compression stocking or crepe bandage
- Drugs—calcium dobesilate 500 mg BD.

#### *Symptomatic uncomplicated cases*

Surgery or endovenous approaches from above can be used as per the vessel involved.

**Indications of surgery**

- Recurrent ulceration despite non-operative management
- Persistent symptoms
- Extremely large varicosities.

**Venous ulcer management**

- It is important to remember that the surgery for varicose veins does not speed up the ulcer healing process and therefore there is no hurry to do surgery till the ulcer heals. Surgery here is indicated only to prevent recurrences and further complications from varicose veins once the ulcer resolves
- '**Bisguard regime**' is the term used to describe the nonoperative management of venous ulcer
- The aim is to convert an infected ulcer to clean ulcer and finally to healing healthy ulcer
- The management steps are the same as above mentioned steps of management of asymptomatic cases with one important addition which is **wound debridement** followed by application of well padded wet to dry dressing and if needed culture specific antibiotics
- Zinc oxide impregnated dressings are said to promote healing and are preferred for dressing venous ulcers. Simple saline and betadine dressings are also helpful and can be used
- The varicosities are addressed by surgery or endovenous approach once the ulcer heals.

**Q23. Enumerate the risk factors for the occurrence of deep vein thrombosis and discuss its management in brief.****Write a note on DVT prophylaxis.**

**Ans.**

**Risk factors for DVT are as follows:**

- **Hypercoaguable states** – **congenital** such as factor V leiden mutation, protein C or S deficiency, Antithrombin III deficiency etc. or **acquired** such as antiphospholipid antibody syndrome, lupus anticoagulant, nephrotic syndrome, etc.
- Old age
- Obesity
- Pregnancy
- Prolonged bed rest due to surgery (postoperative state)
- Past history or family history of venous thromboembolism
- Recent myocardial infarction or stroke
- Hyperhomocysteinemia
- Patients with blood group A are at higher risk of DVT. Patients with blood group O are protected against DVT.

Virchow triad (endothelial injury, hypercoagulability and stasis or turbulent blood flow] explains the pathogenesis of DVT.

**Perioperate DVT prophylaxis**

Given according to Caprini score or Roger score for risk of DVT prophylaxis.



Caprini score	Risk	Prophylaxis
0	Very low (< 0.5%)	None
1, 2	Low (upto 1.5 %)	Mechanical
3, 4	Moderate (upto 3 %)	Mechanical or pharmacologic
5 or more	High (upto 6%)	Both mechanical and pharmacologic
High risk cancer surgery	Very high	Both mechanical and pharmacologic and continue both postoperatively

**Mechanical prophylaxis:** Intermittent pneumatic compression is better than thigh length elastic stockings which is better than knee length elastic stockings.

**Pharmacologic prophylaxis:** Low molecular weight heparin or low dose unfractionated heparin both can be used. However, LMWH enoxaparin is the drug of choice and given in the dose of 1 mg/kg if no renal impairment and 0.5 mg/kg in patients with renal impairment.

**Contraindication to LMWH/LDUFH:** Use Low dose aspirin (160 mg) or Fondaparinux with mandatory mechanical prophylaxis.

#### Management of a case of established DVT

- **Start heparin** 80 units/kg bolus followed by 18 units/kg/hour to maintain aPTT at 60 – 80 seconds/ Factor Xa level 0.3–0.7 units/ml. These factor levels are measured every 6 hours till the patient is on heparin.
- **Also start oral warfarin** to maintain INR 2.5–3 with heparin and stop heparin after overlap of 5–6 days. This is done because warfarin requires this duration for optimal action.
- **Thrombolysis with tissue plasminogen activator or urokinase** is advised if the patient presents within 5-7 days of DVT.
- **Endovascular therapy with balloon dilatation and stenting** can also be done
- **Surgical thrombectomy is indicated only** when patient has Phlegmasia cerulea dolens not responding to medical therapy. It is contraindicated in septic iliofemoral thrombosis cases.
- **Indications of Inferior vena cava filter in these cases are as follows,**
  - Contraindication to anticoagulation therapy
  - Complications due to anticoagulant therapy
  - Floating IVC thrombus
  - Recurrent pulmonary embolism/ Deep vein thrombosis despite anticoagulation therapy.
  - Pulmonary hypertension with recurrent pulmonary embolism

( **ALWAYS REMEMBER:** Phlegmasia alba dolens is deep venous occlusion with patent arterial inflow and collateral venous outflow. Phlegmasia cerulea dolens occurs when along with the deep flow occlusion, the arterial inflow and the collateral venous outflow is also obstructed. Phlegmasia alba dolens precedes phlegmasia cerulea dolens in 50 – 60 % cases.)

**Q24. What is Raynaud phenomenon? Enumerate its causes.**

**Discuss the phases of Raynaud phenomenon and enumerate its causes.**

**Ans.** Raynaud phenomenon is recurrent attacks of episodic triple color response to spasm of digital blood vessels from pallor to cyanoses to rubor also called as blanching to dusky anoxia to red engorgement.

### Pathophysiology

- **Idiopathic** – Raynaud Disease
- **Secondary** – Raynaud syndrome which can be due to any of the following causes
  - Atherosclerosis
  - Burger disease
  - Smoking
  - Thoracic outlet syndrome with vascular involvement
  - Collagen vascular disease [Lupus, rheumatoid arthritis, polyarteritis nodosa, scleroderma]
  - Occupation such as jackhammer workers
  - Multiple myeloma
  - Drugs such as ergot derivatives or beta blockers

### Phases and its cause

The phases begin when the hand is exposed to cold or emotional duress. It is usually seen in females, is bilateral and symmetrical .

Blanching	White	Vasoconstriction
Cyanosis	Blue	Accumulation of deoxygenated blood
Rubor	Red	Resumption of blood supply

Repeated episodes of this kind may result in trophic changes in the affected limbs such as wasting of muscles, loss of subcutaneous fat, ulcers on finger tips, or rest pain and finally gangrene.

**Management** circles on avoidance of the precipitating factors, wearing gloves and socks, removal of cause, smoking cessation, vasodilators such as Nifedipine or ketanserin especially in scleroderma patients, sympathectomy if patient does not feel benefit from other measures.

## MISCELLANEOUS TOPICS IN CTVs

**Q25. Write a note on thoracic outlet syndrome.**

**What is cervical rib? Discuss its management.**

**Ans.** Thoracic outlet syndrome is known by a variety of names such as

- Cervical rib
- Scalenus anticus syndrome
- Costoclavicular syndrome
- Pectoralis minor syndrome
- Hyperabduction syndrome and so on..

The basic anatomy is three different areas through which the axillary vessels and nerves pass to reach the arm.

- These include the region between first rib and the scalenus anterior and medius muscle which can be involved by cervical rib or scalenus anticus syndrome.
- The region between first rib and clavicle – costoclavicular syndrome and
- The region between insertion of pectoralis minor and bones behind – pectoralis minor syndrome.
- **Cervical rib** can be complete, incomplete, fibrous cord or blind ending rib. This may be extra rib from the seventh cervical rib or extra rib from the first thoracic rib.
- **The pathophysiology of symptoms** and the clinical features are due to the involvement of the structures that pass through these three regions which are as follows:
- It is more common in females with long neck and tall stature.

Structure compressed	Symptom	Sign
Subclavian artery	Pain, pallor, poikilothermia, weakness increased by increased work of the affected arm	Trophic changes associated with arterial inflow compromise may be seen. Feeble pulse. Positive Adson test. Weak pulse
Subclavian vein	Heaviness, edema, cyanosis, pain	Edema Dilated veins
Brachial plexus	Pain and paresthesia mainly on the ulnar side of hand and forearm (Ulnar N. m.c. involved) This becomes worse on lifting heavy objects	Wasting of small muscles supplied by ulnar nerve Positive Card test (Egawa) Positive Froment sign
Sympathetic system	Vasomotor changes, Raynaud phenomenon	Raynaud phenomenon can be induced by cold or excessive use of that arm
Other signs	Palpable lump Pulsatile lump	Cervical rib Subclavian aneurysm
Tests	Costoclavicular test (Military position test) Hyperabduction test Adson test Book test, Froment test	Costoclavicular syndrome Hyperabduction test Arterial involvement Ulnar nerve involvement

### Investigations

- X-ray of neck and chest
- MRI for anatomical knowledge of brachial plexus and vessels
- Nerve conduction studies and EMG studies

### Treatment

- Avoidance of heavy weight lifting
- Physiotherapy for muscle strengthening
- Local heat therapy

- Analgesics
- Neurotonic medications such as pregablin, methylcobalamine, histidine containing medications.
- Surgery done when above conservative measures fail and disease keeps progressing despite the conservative measures.
- **Options include**

Cervical rib	Transaxillary or supraclavicular approach to excise the rib completely
Scalenus syndrome	Division of the scalenus insertion
Costoclavicular syndrome	Excision of part of clavicle usually the middle third and part of 1st rib
Pectoralis minor syndrome	Division of the insertion of pectoralis minor
Paget schrotter syndrome [Subclavian vein effort thrombosis]	<b>Acute:</b> Local Tissue plasminogen activator or streptokinase therapy immediately followed by anticoagulation for 3 months. <b>Chronic:</b> Rib resection and followed by revascularisation.

**Q26. Write a note on management of lymphedema of lower limb.**

**Discuss the causes of lower limb lymphedema and outline its management.**

**Ans.**

**Causes of lymphedema are as follows:**

<b>Primary lymphedema</b>	<b>Congenita (Familial form called Milroy disease)</b> Occurs more commonly in males < 2 years old Incidence = 10% <b>Praecox (Familial form called Meig's disease)</b> m.c. type seen in females of 2 – 35 years age group <b>Tarda</b> Seen in people > 35 years old Is rarest of the three.
<b>Secondary lymphedema</b>	Filariases (m.c. cause) Tuberculosis Iatrogenic (Extensive lymph node dissections and excision such as ilioinguinal lymph node dissection) Malignant infiltration of lymph nodes Recurrent infections

**Some syndromes associated with congenital lymphedema** – Turner syndrome, Klinefelter syndrome, Milroy syndrome, Klippel-Trenaunay syndrome.

**Clinical features** can be remembered using Brunner's grading as follows:

**Brunner's grading**

<b>Subclinical</b>	Altered histology of lymphatics but no symptoms
<b>I</b>	Pitting edema
<b>II</b>	Nonpitting edema

*Contd...*

Contd...

III	Skin changes, papillae and fibroses such as <b>Buffalo hump</b> – on dorsum of foot <b>Stammer sign</b> – skin on dorsum of foot cannot be pinched <b>Squaring of toes</b> – occurs due to footwear
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**Complications**

<b>Infections</b>	Recurrent lymphangitis and cellulitis
<b>Malignant transformation</b>	Lymphangiosarcoma

**Investigations**

- **MRI** of the affected limb has now replaced the previously performed lymphangiographic studies.
- **Direct or indirect lymphangiography.**

Direct lymphangiography is the gold standard and is performed by direct instillation of the contrast into the lymphatics whereas indirect lymphangiography is performed using intradermal injection of dye.

**Lymphangiographic classification**

- Congenital hyperplasia – 10%
- Distal obstruction – 80%
- Proximal obstruction with distal dilatation – 10%

**Treatment**

Nonsurgical management is the most important line of management as surgery is doomed by recurrences and complications.

**Nonsurgical management** mainly includes decongestive lymphedema therapy (DLT)

**DLT has 2 phases.****Phase 1** deals with

- Monitored Exercises
- Skin care and foot care
- Manual lymphatic drainage (MLD)
- Multilayer lymphatic bandaging (MLLB)– graded compression bandages with 100 % compression at foot, 70% at knee and 40% at groin. Always measure Ankle brachial pressure index and is  $ABI < 0.5$  than graded compression bandage is not used and replaced by the use of simple compression stockings. Also patients with heart failure or peripheral neuropathies should avoid bandaging and use simple compression garments.

Successful outcome of phase 1 is given when there is no increase in swelling or skin changes.

**Phase 2**

Patient self care regimen with occasional professional intervention and exercises.

**Surgical options***Indications*

- Severe functional disability or disfigurement
- Severe pain and symptoms refractory to nonsurgical management
- Recurrent lymphangitis

<b>Homan operation</b>	Subcutaneous excision followed by skin flap closure
<b>Sistrunk operation</b>	Wedge excision of skin and subcutaneous tissue followed by primary closure
<b>Thompson procedure</b>	Buried one flap under another. Chance of pilonidal sinus formation
<b>Charles operation</b>	Circumferential excision of skin, subcutaneous tissue and fascia followed by graft closure
<b>Kinmoth bypass</b>	Ileal mucosal patch
<b>Neibulowitz bypass</b>	Lymphovenous anastomosis
<b>Gilles operation</b>	Skin bridge operation

**Q27. Enumerate the differences between Hodgkin's and non-Hodgkin's lymphoma. Discuss the management of non-Hodgkin's lymphoma.**

**Classify and discuss the staging of Hodgkin's lymphoma.**

**Enumerate the risk factors for the development of lymphoma and discuss its management.**

**Ans.**

#### **Classification of lymphomas**

<b>Hodgkins lymphoma</b>	<b>Non-Hodgkin's lymphoma</b>
<b>Ryes classification</b> <b>Classic HL</b> Nodular sclerosis Mixed cellularity Lymphocyte depletion Lymphocyte rich – added by REAL Classification group <b>Variant HL</b> Lymphocyte predominant	<b>WHO classification</b> Precursor B cell type Peripheral B cell type Precursor T cell type Peripheral T cell type
Reed Sternberg cell present	No Reed Sternberg cell present
<b>Risk factors</b>	
Bimodal age group – 3rd decade and 6th decade onwards First degree relatives Jewish origin AIDS Epstein Barr virus	<b>Virus</b> – HIV, HHV, HTLV-1 in AIDS or post-transplant patients, HCV, EBV. <b>Bacteria</b> – <i>Borrelia burgdorferi</i> , <i>C.jejuni</i> , <i>C. Psittaci</i> , <i>H.pylori</i> High fat intake in diet Recreational drug use Occupation in forestry
<b>Clinical features</b>	
More often localized to single group of lymph nodes [Cervical/ Mediastinal/ Retroperitoneal]	More often involve multiple peripheral nodes simultaneously
Spread by contiguity and orderly spread, can be predicted.	Unpredictable non-contiguous spread
Extranodal spread is uncommon	Extranodal spread is common
Mesenteric nodes and Waldeyer ring are rarely involved	Mesenteric nodes and Waldeyer ring are commonly involved

Contd...

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Hodgkins lymphoma		Non-Hodgkin's lymphoma
Staging		
Is same for both Hodgkin and Non-Hodgkin lymphoma		
Called <b>modified Ann Arbor staging or Cotswold staging</b>		
I	Single lymph node/extranodal disease site [Spleen, thymus, Waldeyer ring] or single extralymphatic site	
II	2 or more lymph node regions on the same side of diaphragm with/out single ELS involvement	
III	2 or more lymph node regions on both sides of diaphragm with/out single ELS or spleen involvement [III-S] or both [III- ES]	
III-1	Involvement of celiac, portal, splenic nodes	
III-2	Involvement of para-aortic, mesenteric, iliac nodes	
IV	Disseminated involvement of more than one extralymphatic site with/without lymph node involvement or Bone marrow or liver involvement.	
A	No symptoms	
B	Fever [ $> 38^{\circ}\text{C}$ ], Night sweats, weight loss $> 10\%$ in $< 6$ months	
X	Bulky disease $> 10\text{ cm}$ or more than $1/3^{\text{rd}}$ widening of mediastinum	
E	<b>Extranodal site [ELS]</b> proximal or contiguous to the primary site	
Treatment		
<b>Stage I, II</b> Treatment of choice is chemotherapy 2-3 cycles followed by radiotherapy to involved sites <b>All other Stages</b> 8 cycles chemotherapy <b>Regimen</b> <b>ABVD</b> [Adriamycin, Bleomycin, Vinblastine, Dacarbazine] <b>MOPP</b> [Mechlorethamine, Vincristine, Prednisone, Procarbazine] <b>BEACOPP</b> <b>Stanford V regime</b>		NHL of different sites have different management options from chemotherapy, radiotherapy or surgery or combination of these therapy options Chemotherapy regime in this variety of lymphoma include <b>CHOP</b> <b>CHOP + Rituximab</b>

**Q28. Enumerate the causes of unilateral lower limb edema.****Ans.** Causes of unilateral lower limb edema are as follows

<b>Lymphedema</b>	<ul style="list-style-type: none"> <li>Discussed below</li> </ul>
<b>Venous disorder</b>	<ul style="list-style-type: none"> <li>Varicose veins</li> <li>Deep vein thrombosis</li> <li>Inferior vena cava thrombosis</li> </ul>
<b>Infection</b>	<ul style="list-style-type: none"> <li>Cellulitis</li> <li>Abscess</li> </ul>
<b>Others</b>	<ul style="list-style-type: none"> <li>Adiposes dolorosa</li> <li>Arteriovenous fistula</li> <li>Erythrocyanosis</li> <li>Extensive burns</li> <li>Fractures</li> </ul>

**Q29. Enumerate the causes of non-healing ulcer of lower limb and discuss its management.**

**Enumerate the causes of ulcer of lower limb and discuss the management of a non-healing ulcer of lower limb.**

**Ans.**

**Causes of lower limb ulcer**

<b>Arterial</b>	<ul style="list-style-type: none"> <li>• All causes of peripheral arterial disease</li> </ul>
<b>Venous</b>	<ul style="list-style-type: none"> <li>• All causes of varicose veins and DVT</li> </ul>
<b>Neuropathic</b>	<ul style="list-style-type: none"> <li>• Syringomyelia</li> <li>• Tabes dorsalis</li> <li>• Hemiplegia</li> <li>• Paraplegia</li> </ul>
<b>Diabetic</b>	
<b>Infective</b>	<ul style="list-style-type: none"> <li>• Tuberculosis</li> <li>• Syphilitic</li> <li>• Other infections</li> </ul>
<b>Neoplastic</b>	<ul style="list-style-type: none"> <li>• Squamous cell cancer</li> <li>• Basal cell cancer</li> </ul>
<b>Traumatic ulcers</b>	
<b>Other causes</b>	<ul style="list-style-type: none"> <li>• Tropical ulcer</li> <li>• Poliomyelitis</li> <li>• Sickle cell disease</li> <li>• Chronic lymphedema</li> </ul>

**Causes of non-healing ulcer of lower limb**

<b>Arterial</b>	<ul style="list-style-type: none"> <li>• All causes of peripheral arterial disease</li> </ul>
<b>Venous</b>	<ul style="list-style-type: none"> <li>• All causes of varicose veins and DVT</li> </ul>
<b>Bony cause</b>	<ul style="list-style-type: none"> <li>• Osteomyelitis</li> </ul>
<b>Infective</b>	<ul style="list-style-type: none"> <li>• Buruli ulcer / Bairnsdale ulcer [<i>M. fortuitum</i> or <i>M. ulcerans</i>]</li> <li>• Actinomycoses</li> <li>• Meliodoses</li> <li>• MRSA</li> <li>• Other nontubercular mycobacteria</li> </ul>
<b>Malnutrition and immunosuppression</b>	<ul style="list-style-type: none"> <li>• Anemia, Hypoproteinemia</li> <li>• AIDS, Chemotherapy patients etc.</li> </ul>
<b>Vasculitis</b>	<ul style="list-style-type: none"> <li>• Lupus</li> <li>• Rheumatoid arthritis</li> <li>• Polyarteritis nodosa</li> </ul>
<b>Diabetes</b>	
<b>Neuropathic</b>	<ul style="list-style-type: none"> <li>• Syringomyelia</li> <li>• Tabes dorsalis</li> <li>• Hemiplegia</li> <li>• Paraplegia</li> </ul>
<b>Skin malignancy</b>	<ul style="list-style-type: none"> <li>• Squamous cell cancer</li> <li>• Basal cell cancer</li> </ul>



**Management outline**

- Ulcer edge biopsy
- Bone biopsy
- Chest X-ray and Mantoux testing
- X-rays and MRI of the involved limb
- Duplex imaging of the involved limb
- All cultures—wound, blood, urine

**Treatment**

- Take care of general condition of the patient and improve the nutritional status and immune condition of the patient
- Treatment of the underlying cause
- Convert the ulcer from infected to clean to healing ulcer with serial debridements and dressings.
- Appropriate antibiotic therapy if the patient has any infection.
- If all the causes are ruled out and ulcer is still nonhealing—treat with biological dressings or with excision and skin grafting.

# SECTION

# 6

## **Breast and Endocrine Surgery**

- Breast
- Thyroid
- Parathyroid and Adrenal



## BREAST

### Q1. Write a note on blood supply and lymphatic drainage of breast.

#### Discuss the anatomy of breast.

**Ans.** Breasts are modified sweat glands.

#### Embryology

The breasts develop along the milk-line that extends between the limb buds from the axilla to the inguinal region distally.

#### Location

The breasts are located within the superficial fascia of the anterior chest wall.

#### Extent

- Superiorly second or third rib.
- Inferiorly the inframammary fold that is located at the level of the sixth or seventh rib.
- Medially the lateral border of the sternum.
- Laterally to the anterior or midaxillary line.
- The posterior or deep surfaces of the breast rest upon portions of the fasciae of the pectoralis major, serratus anterior, external oblique muscles and upper portions of the anterior rectus sheath.
- Breast tissue extends commonly into the anterior axillary fold as the *axillary tail of Spence*.
- The upper half of the breast, particularly the upper outer quadrant, contains the greater volume of glandular tissue than the remainder of the breast.

#### Structure

- 15 to 20 lobes of tubuloalveolar glandular tissue,
- Fibrous connective tissue that supports its lobes, and
- Adipose tissue that resides in parenchyma between the lobes.

#### Subcutaneous connective tissue

It surrounds the gland and extends as septa between the lobes and lobules which connect the deep layer of superficial fascia to dermis of skin, providing support to the gland. These are **the Cooper's ligaments**.

#### Superficial fascia

The deep layers of superficial fascia on the posterior surface of the breast fuse with the deep (pectoral) fascia of the chest wall.

#### The retromammary space

A space between the deep layer of the superficial fascia and the deep investing fascia of the pectoralis major and the contiguous muscles of the thoracic wall. It contributes to the mobility of the breast on the chest wall.

**Arterial supply**

- Lateral branch of posterior intercostal artery
- Perforators from internal mammary artery which arborize with 2nd, 3rd and 4th anterior intercostal perforators to form **medial mammary artery**.
- Branches of axillary artery
  - Highest thoracic artery
  - Lateral thoracic artery (called **lateral mammary artery**)
  - Pectoral branch of thoracoacromial artery.

**Venous drainage**

- Perforating branches to internal thoracic vein.
- Perforating branches to posterior intercostal vein.
- Tributaries to axillary vein.
- Tributaries to Batson's vertebral venous plexus.

**Nerve supply**

- Lateral cutaneous branches of 3rd to 6th intercostal nerves.
- Anterior branch of supraclavicular nerve.

**Lymphatics**

- Dermal lymphatics drain to subdermal lymphatics to subareolar plexus of Sappey to lymph nodes.
- Lymph nodes groups
  - **Anterior group** (external mammary or pectoral group)—along the lateral thoracic vessels
  - **Posterior group** (subscapular or scapular group)
  - **Lateral group** (axillary vein group).
- These three groups comprise the **level 1 nodes** and they all drain into central group.
- Some lymphatics drain directly from the breast into central nodes
  - **Rotter group** (Interpectoral group or **level II nodes**) is located between pectoralis major and minor and also drains into the **central group**
  - The central group as well as some branches from breast and Rotter group drain into the **apical group (subclavicular group)**
  - The apical group finally drains into the **supraclavicular group**.

**Q2. What are the causes of nipple discharge? Discuss its management.**

**Ans.** Nipple discharge is a common symptom and needs to be evaluated frequently in surgery clinics.

The important first step is to do history and physical examination to look for the type of nipple discharge and see whether a lump is palpable or not. The risk of malignancy increases if there is an underlying mass.

**Causes of nipple discharge are as follows:**

	Type of discharge	With lump	Without lump
<b>Discharge from single duct</b>	Serous discharge	Duct ectasia Fibrocystic disease Rarely malignancy	Duct ectasia Fibrocystic disease Rarely malignancy
	Blood-stained	Intraductal papilloma (mc) Intraductal carcinoma Duct ectasia	Intraductal papilloma (mc) Intraductal carcinoma Duct ectasia
<b>Discharge from multiple ducts</b>	Milk	Hyperprolactinemia (Pituitary microadenoma {Prolactinoma} is mc) Lactation Hypothyroidism	Galactocele
	Serous	As above	As above
	Blood stained	As above	As above
	Purulent	Breast abscess Periductal mastitis	
	Grumous/ Greenish	Duct ectasia	Duct ectasia
<b>Discharge from breast surface</b>	Bilateral	Eczema, psoriasis	
	Unilateral	Paget's disease	Paget's disease

**Assessment***History*

- Unilateral or bilateral
- Duration, frequency, amount and type of nipple discharge
- Whether it occurs spontaneously or only on squeezing
- Other symptoms—lump or pain.

*Examination*

- Lump, single duct or multiple duct, tenderness and other signs of inflammation
- Nipple and areola examination for inversion, recent retraction, eczema, excoriation or discharge
- Axilla and supraclavicular region for lymph nodes always to be examined.

***“Single duct, spontaneous, unilateral, blood-stained, clear or serous in a women above 40 years especially with a mass is suggestive of malignancy and needs evaluation.”***

**Investigations**

- Mammogram for women >35 years and for any age when with lump.
- USG breast for younger women without lump.
- FNAC and/or trucut from the lump, if present.

Cytology of the nipple discharge is rarely of help.

- **If patient has profuse bilateral milky discharge**, rule out pregnancy and measure serum prolactin and thyroid function test

- **Punch biopsy** to rule out Paget's disease and for any unexplained nipple eczema or ulceration. If still suspicious but false result, reassess and consider for open biopsy.
- If patient has high-risk features and none of the above studies reveals a thing than a **retroareolar open biopsy or microdochectomy with excision of mass, if present may be necessary.**
- Ductography and discharge cytology have not much significance because whatever be the result, if other tests are inconclusive, microdochectomy or retroareolar open biopsy and excision of any mass, if present, as such are going to be performed. So they do not add anything to the management and are, therefore, not performed routinely.

**Management depends on the cause of the nipple discharge.**

**Q3. What is ANDI (aberrations of normal development and involution)? Discuss its types and management in brief.**

**Ans.** ANDI encompasses all aspects of breast condition including pathogenesis and various degrees of abnormalities.

**Spectrum of ANDI is as follows:**

Age	Normal development	Disorder	Disease
<b>Early reproductive years (15–25 years)</b>	Lobular development Stromal development Nipple eversion	Fibroadenoma Adolescent hypertrophy Nipple inversion	Giant fibroadenoma Gigantomastia Subareolar abscess/ mammary duct fistula
<b>Late reproductive years (25–40 years)</b>	Cyclical menstruation Epithelial hyperplasia	Cyclical mastalgia/ nodularity Bloody nipple discharge	Incapacitating mastalgia
<b>Involution (35–55 years)</b>	Lobular involution Ductal involution Dilatation Sclerosis Epithelial turnover	Macrocysts Sclerosing lesions Duct ectasia Nipple retraction Epithelial hyperplasia	Periductal mastitis Epithelial hyperplasia with atypia

Risk of malignancy in these entities is explained according to the **Page Dupont categories** which again classify ANDI and give risk as follows:

Lesion	Types of disease	Relative risk
<b>Nonproliferative changes</b> (Fibrocystic disease also called Reclus disease/Schimmelbusch disease/Cooper's disease/mazoplasia/cystic mastopathy)	Adenosis Duct ectasia Mild epithelial hyperplasia	No risk of malignancy
<b>Proliferative disease without atypia</b>	Moderate or florid hyperplasia Sclerosing adenosis Papilloma	1.5–2 fold increased risk
<b>Proliferative disease with atypia</b>	Atypical ductal hyperplasia Atypical lobular hyperplasia	4-fold increased risk

**Management**

- Simple fibroadenomas with confirmed diagnoses (FNAC) need only reassurance and follow-up. Excision is reserved when diagnoses is in suspicion.

- Duct ectasia is managed by microdochotomy/Hadfield operation and is discussed separately.
- Nipple discharge and mastalgia management are discussed separately.

**Q4. Write a note on mammary duct ectasia.**

**What is Zuska disease? Discuss its management.**

**Ans.**

**Definition**

It is dilatation of lactiferous ducts with or without periductal inflammation, therefore, also known as **periductal mastitis**.

**Pathogenesis**

Obscure, more common in smokers.

**Pathology**

- Dilatation of duct is followed by filling of the ducts with brown or green fluid which produces the nipple discharge.
- These fluids stimulate the inflammatory reaction and lead to periductal mastitis.
- If not checked at this stage, it can also lead to abscess or mammary duct fistula formation.
- Chronic mastitis can lead to fibrosis and result in recent onset nipple retraction.
- This chronic mastitis and associated mass can mimic carcinoma.

**Clinical features**

It can present as any type of nipple discharge, or as mammary duct fistula, abscess or nipple retraction.

**Management**

- Diagnoses is done as explained in the question on nipple discharge management.
- Antibiotics are started once the diagnoses is established.
- Cessation of smoking.
- **Hadfield operation (Radical duct excision)** is the operation of choice.

**Recurrent periductal mastitis** is called **Zuska's disease**. Management is with incision and drainage, antibiotics and cessation of smoking.

**Q5. What is phylloides tumor? Discuss its clinical features and management.**

**Ans.**

- It is also known as serocystic disease of Brodie.
- It is the most common breast sarcoma.

**Age**

Around 40 years.

**Pathology**

- It is a tumor of monoclonal origin with hypercellularity and consists of both stromal and epithelial components.
- Gross: Well-defined borders, cut surface shows leaf-like appearance, therefore, called phylloids tumor.



- It can be benign, borderline or malignant. Malignancy can be low grade or high grade.
- **Stromal:** Lipomatous, sarcomatous, rhabdomyosarcomatous elements can be present. Stroma forms the bulk of the tumor.
- **Epithelial:** It is less in amount than the stromal elements.
- **Metastasis:** Hematogenous to lung, bone, mediastinum and abdominal viscera.

#### Clinical features

- The tumor presents in middle-aged females.
- It is well-defined, mobile over chest wall, can assume large sizes with dilated vessels over the lump and well-defined margins. Surface is smooth or bosselated. Consistency is firm to variegated and rubbery.
- It is usually nontender and not fixed to skin. There is no lymphadenopathy.

#### Differential diagnoses

**Malignancy:** No lymphadenopathy, nonfixity to skin and chest wall despite the large size, no nipple and areola changes.

#### Management

- Diagnoses is using the same investigations that are used for breast lump, **i.e. triple examination of mammogram/USG, FNAC and trucut biopsy.**
- **Treatment** is by simple excision with 1–2 cm margin for small tumors and simple mastectomy for large tumors or malignant phylloides.
- **Axillary dissection is not required as it does not spread through lymphatics.**

#### Q6. Write a note on Paget's disease of nipple.

Ans.

- Paget's disease of nipple is a cutaneous marker of underlying malignancy either invasive or in situ ductal lesion.
- It is unilateral and rarely bilateral chronic eczematous eruption of nipple which can progress to ulcerated weeping lesion.
- The underlying lesion may be palpable or nonpalpable.

#### Pathology

**Paget cell:** Large cell, pale staining, with round nuclei and large nucleoli.

They migrate from the nipple to lactiferous sinus to underlying breast epithelium. But never invade the basement membrane on their own.

#### Clinical features

- Present as unilateral/ bilateral eczema, nipple discharge, with/without lump and/or lymph nodes.
- Nearly all patients have underlying malignancy and more so when Paget's is associated with a breast lump.

#### Differential diagnoses

- **Eczema:** It is bilateral. No underlying lump is present in eczema.
- **Superficial spreading melanoma:** Paget's is CEA positive whereas melanoma is S100 positive.

**Management**

- Mammogram/USG, biopsy to establish the diagnoses.
- Treatment is with simple mastectomy. Further management depends on the histopathology report.

**Q7. Write a note on causes of gynecomastia.****What is gynecomastia? Discuss its causes.****Ans. Causes of gynecomastia are as follows:***Physiological*

- Neonatal gynecomastia due to placental estrogens.
- Adolescent gynecomastia due to relative estrogen excess.
- Senescent gynecomastia due to relative testosterone deficiency.

*Pathological*

- Idiopathic—mc
- Estrogen excess

Absolute	Relative
<ul style="list-style-type: none"> <li>• <b>Increased testicular production</b> <ul style="list-style-type: none"> <li>– Testicular tumors (Leydig cell, sertoli cell, granulosa/ theca cell tumor)</li> <li>– Bronchogenic carcinoma and transitional cell tumor of urinary tract</li> </ul> </li> <li>• <b>Increased aromatization</b> <ul style="list-style-type: none"> <li>– Adrenal hyperplasia or carcinoma</li> <li>– Cirrhoses, thyrotoxicoses, exogenous androgen administration</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• <b>Primary testicular failure</b> <ul style="list-style-type: none"> <li>– Anorchia, Klinefelter syndrome, testicular feminization syndrome</li> </ul> </li> <li>• <b>Secondary testicular failure</b> <ul style="list-style-type: none"> <li>– Orchitis, trauma, castration, leprosy,</li> <li>– Renal failure</li> <li>– Myotonic dystrophy or spinal cord injury</li> </ul> </li> </ul>

- **Common Drugs (DOC<sup>4</sup>KS )**

Digitalis, oral contraceptive pills, cimetidine, clomiphene, captopril, calcium channel blockers, ketoconazole, spironolactone.

**Other drugs:** Isoniazid, tricyclic antidepressants, methyldopa, flutamide.

**Simon grading**

<b>Grade 1</b>	Mild enlargement, no skin redundancy
<b>Grade 2A</b>	Moderate enlargement, no skin redundancy
<b>Grade 2B</b>	Moderate enlargement, skin redundancy
<b>Grade 3</b>	Marked enlargement with skin redundancy and ptosis

**Investigations**

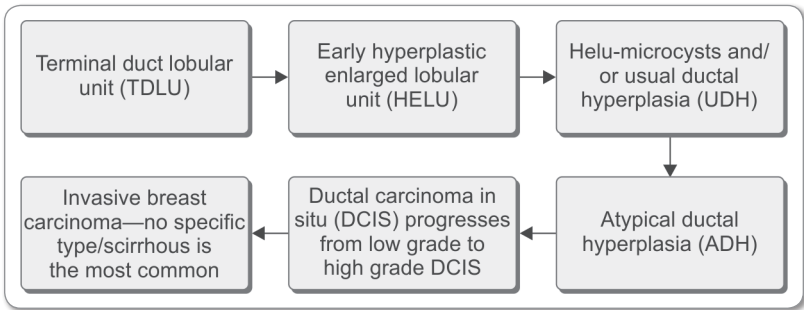
- **History and physical examination.**
- **Evaluate testis:** Testicular ultrasound, serum testosterone, LH, DHEAS, endocrine profile—estrogen, prolactin, adrenal CT.
- **Thyroid function tests.**
- **Breast** mammogram, ultrasound, biopsy.
- Liver function test, abdominal CT.

**Treatment**

- Stop offending drug.
- Treat the systemic disease, if present.
- Karyotyping for klinefelter if positive, consider bilateral mastectomy.
- Most cases resolve spontaneously and 1 year observation period is suggested.
- Pharmacology during observation: Tamoxifen, danazol, aromatase inhibitors all have been used in the treatment of gynecomastia.
- Surgery is done for gynecomastia of long duration, cosmetic or psychological reason, symptomatic or suspected malignancy.
- Simple mastectomy, subcutaneous mastectomy, liposuction, reduction mammoplasty are all suggested procedures.

**Q8. Write a note on evolution/natural history of breast cancer.**

**Ans.** Natural history of breast cancer is explained by modified Wellings Jensen model which is as follows:



**Q9. Differentiate between LCIS and DCIS.**

**Ans.**

LCIS (Lobular carcinoma in situ)	DCIS (Ductal carcinoma in situ)
<b>Pathology</b>	
Growth occurs in terminal duct lobular unit	Growth occurs within the duct lumen
Characteristic histological picture – Indian file pattern- linear strands of tumor cells	Can be low grade (micropapillary/papillary/cribriform) or high grade (comedo and solid types)
<b>Clinical features</b>	
Age-44–48 years	Age-54–58 years
Incidence 2–5%	Incidence 5–10%
Occur only in females	5% incidence in males also.
Usually incidental finding with no symptoms	Can present with nipple discharge, mass, mastalgia
Multicentricity and bilaterality more common	Less common
<b>Risk of malignancy</b>	
Synchronous carcinoma in 2–5%	Synchronous carcinoma in 2–46%
Risk of malignancy in both breasts	Risk of malignancy only in ipsilateral breast

LCIS (Lobular carcinoma in situ)	DCIS (Ductal carcinoma in situ)
Metastasis is more common to peritoneum and meninges	Metastases is more common to lung, liver, bone
Relative risk of malignancy is increased by 10 fold in both the types of in-situ cancers Invasive ductal carcinoma is the most common malignancy to arise in both the types	
Radiology	
Show “neighborhood calcification” in mammogram	Show “microcalcification” in mammogram (mc presentation in 90% cases)
Almost never has mass	Carcinoma have a malignant mass
Management	
Observation/ chemoprevention/ prophylactic bilateral mastectomy	Low grade—lumpectomy to negative margins Limited disease—lumpectomy + radiotherapy >4 cm/multicentric/multifocal disease—mastectomy
Negative microscopic margin, size and grade has no bearing on prognosis	Van Nuys prognostic index (age, margin, grade, and size) is used for these patients

**Q10. Write the staging of breast cancer.****Ans. AJCC 7th edition staging is as follows:***T stage*

- Tx – primary cannot be assessed.
- T0 – no primary tumor.
- T (is) – can be DCIS/LCIS/Paget’s.
- T1 - < or = 2 cm (a- > 1 to < 6mm, b – 6 mm to < 11 mm, c – 11 mm to < or = 20 mm).
- T2 - >2 cm to < or = 5 cm.
- T3 - >5 cm.
- T4a – chest wall involvement.
- T4b – ulceration/ satellite nodules/ peau d’orange appearance.
- T4c – Both T4a and T4b.
- T4d – inflammatory breast cancer—clinicopathologic entity characterized by edema and erythema (with/without peau d orange appearance) involving 1/3 or more of the breast.

*N stage*

- Nx – cannot be assessed.
- N0 – no nodes palpable.
- N1 – metastasis to mobile level I, II axillary nodes.
- N2a – metastasis to matted level I, II axillary nodes.
- N2b – metastasis to internal mammary nodes in absence of axillary metastasis.
- N3a – metastasis to infraclavicular nodes.
- N3b – metastasis to both internal mammary and axillary nodes.
- N3c – metastasis to supraclavicular nodes.

*M stage*

- M0 – no metastasis.
- M1 – metastasis present.

**Staging**

- **Stage 1** – T1N0M0.
- **Stage 2A** – T2N0M0, **T0N1M0**, T1N1M0.
- **Stage 2B** – T3N0M0, T2N1M0.
- **Stage 3A (N2)** – T1-3N2M0, T3N1-2M0, **T0N2M0**.
- **Stage 3B (T4)** – T4N0 – 2M0.
- **Stage 3C (N3)** – any T N3 M0.
- **Stage 4 (M1)** – any T, any N, M1.

Stage 1 and 2 A, B are called **Early breast cancer**, whereas, stage 3A, B, C is called **Locally advanced breast cancer**.

**Q11. What is triple breast assessment? Discuss its role in breast cancer management.**

**Ans. Triple breast assessment includes:**

- Clinical breast examination.
- Imaging (USG or mammography).
- Tissue sampling (FNAC or true cut biopsy).

**Imaging details are given in radiology section.**

**Clinical breast examination** should be done 3 yearly from age 20 years and yearly from age of 40 years supplemented with self breast examination monthly started from age 20 years.

**Points on tissue sampling**

FNAC	True cut biopsy
<ul style="list-style-type: none"> <li>• It is easily performed, is rapid, inexpensive, and no incision is required</li> <li>• It requires a trained cytopathologist to report</li> <li>• Cannot distinguish between in situ and invasive cancer</li> <li>• Markers (ER, PR, Her-2-neu) are not routinely available</li> </ul>	<ul style="list-style-type: none"> <li>• This also does not need any incision, and can be read by any cytopathologist with basic training</li> <li>• Markers can be performed on this specimen</li> <li>• It reliably distinguishes in situ and invasive carcinoma</li> <li>• Technique of choice in patients who are planned to receive preoperative systemic therapy</li> </ul>

Triple assessment is the method of choice because all three together increase the diagnostic accuracy to 99.9%. Either of them alone is not as sensitive and also has false negatives. Thus, triple assessment is preferred.

**Q12. Enumerate the risk factors for breast cancer.**

**Ans.** The risk factors for breast cancer are as follows:

- **Age:** Incidence increases with age.
- **Sex:** More common in females.
- **Ethnicity:** More common in jews.
- **Residence:** More common in western countries in high socioeconomic status
- **Personal history:** Combined hormone replacement therapy (estrogen alone does not increase risk), alcohol, high fat diet.

- **Family history:** BRCA1, 2 for familial breast cancer (20–30%) and LiFraumeni syndrome, ataxia telangiectasia, HNPCC, Peutz Jegher syndrome, Cowden syndrome, Muir Torre syndrome, BRCA1, 2 for hereditary breast cancer (5–10%)
- **Past history:** Radiation exposure, breast biopsies and benign breast disease, history of ovarian, endometrial or contralateral breast cancer in females and history of prostate cancer in males.
- **Menstrual history:** Early menarche, late menopause, late first full-term pregnancy (after 30 years), nulliparity, obesity.

#### Models to calculate the risk from risk factors

Gail model (commonly used)	Claus model
<b>Points included</b> <ul style="list-style-type: none"> <li>• Age at menarche</li> <li>• Age at first live birth</li> <li>• Number of first degree relatives with breast cancer</li> <li>• Number of breast biopsies</li> </ul>	<b>Points included</b> <ul style="list-style-type: none"> <li>• First and second degree relatives with breast cancer</li> <li>• Their age at diagnoses</li> </ul>

#### Q13. Outline the management of early breast cancer.

**Write a note on breast conservative surgery.**

**How do we manage axilla in patients with breast conservative surgery?**

**What is the role of various modalities of treatment for a patient with Stage IIA breast cancer?**

**Ans. Early breast cancer:** Stage I, stage IIA and IIB.

**Investigations** for a patient with early breast cancer are as follows:

- Mammography of both breasts.
- USG breast if patient is younger than 30 years.
- FNAC and trucut biopsy of the lump with hormonal status (ER, PR, Her2/neu amongst others).
- USG abdomen and pelvis.
- Chest X-ray.
- Liver function test, ECG and ECHO as patient will be given adriamycin-based chemotherapy.
- Skeletal survey (X-rays of axial skeleton and skull).
- If patient is symptomatic for suspicious metastasis than upgrade the metastatic workup to include CECT abdomen and chest, NCCT head, as well as a bone scan.
- Routine preanesthetic work up to include complete blood count, coagulation profile, serum protein levels, kidney function, electrolytes and sugar levels.

#### Treatment

Paradigm has now shifted toward breast conservative surgery for most patients of early breast cancer if they do not have the contraindications.

#### Contraindications of BCT:

- **Absolute**
  - Pregnancy

- History of therapeutic irradiation to chest
- Persistent positive margins after repeated surgical attempts
- Diffuse microcalcifications in 2 or more quadrants
- 2 or more lumps in more than 1 breast quadrant.
- **Relative**
  - History of collagen vascular diseases (scleroderma/ lupus)
  - Large pendulous breast as it interferes with radiation and small breast containing large lump, preclude BCT.
  - Central tumor for which nipple areolae need to be removed for oncologic clearance.

### **Procedure**

- Quadrantectomy, axillary dissection and radiation (QUARTZ).
- Lumpectomy + postoperative radiation.
- Wide local excision (most preferred) + postoperative radiation.

### **Management of axilla in BCT**

**Aim:** To stage, to treat and to prognosticate.

**Procedures:** Sentinel lymph node biopsy, axillary sampling, axillary dissection.

#### *SLNB*

- **Indications:** All patients undergoing BCT with N0 axilla.
- **Contraindications:** Prior axillary surgery, chemotherapy/radiotherapy, palpable lymphadenopathy, multifocal breast cancer.
- **Technique:**
  - **Step 1:** On day prior to surgery, inject 0.2 – 0.5 mL of 0.5 mCi of 0.2 micron radiolabeled technetium 99 sulfur colloid at cancer site and subdermally at 3–4 sites.
  - **Step 2:** In OT, 4 mL of isosulfan blue dye is injected similarly with 1 mL in between tumor and skin. Node is now identified using gamma camera and after incision by following blue afferent lymph vessels to the lymph node.
  - **Step 3:** The identified lymph node is sent for frozen section examination.
- **Complications**
  - Hematoma, seroma, wound infection
  - Lymphedema (4%)
  - Allergic reaction
  - Inability to identify lymph node (10%).

#### *Axillary dissection*

- **Indications:**
  - Preoperative diagnoses of nodal metastasis
  - Positive/ failed sentinel lymph node dissection
  - Nonavailability of SLNB.
- **Incision:** Lazy 's'/skin crease/anterior axillary fold incision and then axilla dissected as in modified radical mastectomy.

Complications are more common with axillary dissection and incidence of lymphedema increases to around 25–30%.

*Indications of adjuvant chemotherapy (systemic control of disease)*

- Tumor size >0.5 cm with high grade tumor, lymphovascular invasion, hormone receptor negative or Her2/neu positive status.
- Lymph node positivity.
- Tumor size >1 cm.

Cyclophosphamide, adriamycin, 5-fluorouracil or paclitaxel-adriamycin combinations are routinely used for 6 cycles.

*Indications of adjuvant radiotherapy (local control of disease)*

- All patients with BCS require adjuvant radiation therapy.

*Indications of adjuvant hormone therapy*

- All patients with tumor size >1 cm should be given hormone therapy.

Tamoxifen in premenopausal and aromatase inhibitors in post-menopausal age for a duration of 5 years.

**Prognosis**

- St. Gallen's prognostic index.
- **Nottingham prognostic index** =  $(0.2 \times \text{tumor size in cm}) + \text{tumor grade (1-3)} + \text{lymph node stage (1-3)}$ 
  - Value < or = 2.4 – excellent prognosis
  - Value < or = 3.4 – good prognosis
  - Value < or = 5.4 – moderate prognosis
  - Value > 5.4 – poor prognosis.

**Follow-up**

- Monthly self examination
- 6 monthly clinical examination and systemic examination for 1st 2 years and yearly thereafter.
- Yearly mammogram.
- Metastatic follow-up as per the symptoms.

**Q14. Outline the management of locally advanced breast cancer.****Write a note on types of mastectomy.****How do we manage patients with stage IIIA breast cancer?****What is the role of various modalities of treatment for a patient with locally advanced breast cancer?**

**Ans. Locally advanced breast cancer:** Stage IIIA, stage IIIB and IIIC.

**Investigations** for a patient with early breast cancer are as follows:

- Mammography of both breasts.
- USG breast if patient is younger than 30 years.
- FNAC and trucut biopsy of the lump with hormonal status (ER, PR, Her2/neu amongst others).
- CECT chest and abdomen and pelvis.
- Liver function test, ECG and ECHO as patient will be given adriamycin-based chemotherapy.



- Bone scan.
- NCCT head.
- Routine preanesthetic workup to include complete blood count, coagulation profile, serum protein levels, kidney function, electrolytes and sugar levels.

### Treatment

Some patients of LABC are being offered breast conservation therapy now but standard treatment for stage III (LABC) is still **neoadjuvant chemotherapy** (3 cycles of same regime as in adjuvant setting with or without hormone therapy) **followed by modified radical mastectomy followed by adjuvant chemotherapy + radiotherapy + hormonal therapy.**

### Types of mastectomy

- Extended radical mastectomy: Radical + internal mammary nodes + supraclavicular and upper mediastinal nodes.
- Super radical mastectomy: Radical + supraclavicular and upper mediastinal nodes.
- Radical mastectomy: Halstead.
- Modified radical mastectomy
  - **Patey and Dyson:** Remove pectoralis minor and clear level I, II, III nodes
  - **Scanlon:** Divide pectoralis minor to remove level I, II, III nodes
  - **Madden and Auchincloss:** Preserve pectoralis minor and do not remove level III nodes.

Amongst these, Auchincloss modified radical mastectomy is the most commonly performed procedure nowadays.

#### *Indications of adjuvant chemotherapy (systemic control of disease)*

- Tumor size >0.5 cm with high grade tumor, lymphovascular invasion, hormone receptor negative or Her2/neu positive status.
- Lymph node positivity.
- Tumor size >1 cm.

Cyclophosphamide, adriamycin, 5-fluorouracil or paclitaxel-adriamycin combination are routinely used for 6 cycles.

#### *Indications of adjuvant radiotherapy (local control of disease)*

- All patients with LABC require adjuvant radiation therapy.
- All patients with node positivity >4.
- Margin positive patients of modified radical mastectomy.
- All patients offered BCT.

#### *Indications of adjuvant hormone therapy (For hormon receptor positive patients)*

- All patients with tumor size >1 cm should be given hormone therapy.
- Tamoxifen in premenopausal and aromatase inhibitors in postmenopausal age for a duration of 5 years in ER<sup>+</sup>/PR<sup>+</sup> patients and Herceptin in Her-2-neu<sup>+</sup> patients.
- Other options—bilateral oophorectomy, anti-estrogens (fulvestrant), LHRH analogues (Goserelin, buserelin etc), progestins.

### Prognosis

- **St. Gallen's prognostic index**

- **Nottingham prognostic index** =  $(0.2 \times \text{tumor size in cm}) + \text{tumor grade (1-3)} + \text{lymph node stage (1-3)}$ 
  - Value  $< \text{or} = 2.4$  – excellent prognosis
  - Value  $< \text{or} = 3.4$  – good prognosis
  - Value  $< \text{or} = 5.4$  – moderate prognosis
  - Value  $> 5.4$  – poor prognosis.

#### Follow-up

- Monthly self-examination
- 6 monthly clinical examination and systemic examination for 1st 2 years and yearly thereafter.
- Yearly mammogram.
- Metastatic follow-up as per the symptoms.

### Q15. Enumerate the treatment options for breast reconstruction.

#### Ans. Principles of reconstruction in cancer surgery

- Oncologic clearance.
- Functional restoration.
- Cosmesis.

#### Why reconstruction

- Psychological benefit.
- Eliminates constant reminder of their disease.
- Alleviates feeling of deformity.

#### Objectives of breast reconstruction

- Obtain symmetry with the opposite breast.
- The preservation or restoration of the inframammary fold.
- Obtaining satisfactory projection.
- Obtaining satisfactory and matching ptosis with the contralateral breast.

#### Patient selection

- **The risk factor severity score devised by Carl Hartrampf** can be applied to most patients, regardless of the technique used.

#### More than 3 of the following risk factors confer a risk of failure of any reconstructive procedure used:

- Smoking/substance abuse.
- Diabetes.
- Concomitant disease (cardiac/pulmonary).
- Obesity.
- Psychological/Emotional status of the patient.

#### Immediate reconstruction

##### Benefits

- Avoids the psychological trauma of an absent breast.

- Significant gains in emotional well-being, vitality, general mental health, and social functioning.
- Decreases the number of required procedures.
- Decreasing the cost.
- Reducing the risk of multiple exposures to anesthesia.

#### *Concerns*

- Concern of margin positivity—role of frozen section.
- Postoperative radiation.
- Delay in administration of chemotherapy.

Delayed reconstruction has a lower rate of infection and allows time to modify the surgical method to account for radiation therapy. Immediate reconstruction compared with delayed or no reconstruction, reduces psychiatric morbidity reported three months post-operatively and is, therefore, preferred.

### **Options for breast reconstruction**

#### *Autogenous*

- TRAM.
- Deep inferior epigastric perforator flap.
- Latissimus dorsi musculocutaneous flap.
- *Free flaps*
  - Gluteal flap.
  - Superiorly based.
  - Inferiorly based.

#### *Alloplastic*

- Silicone gel implant.
- Silicone implant with saline fill.
- Smooth wall.
- Textured wall.
- Round.
- Anatomic-shaped.

### **Combination procedures**

- Latissimus dorsi flap with implant.
- TRAM flap with implant.

### **Implant reconstruction**

#### *Indications*

- Bilateral reconstruction.
- Patient requesting augmentation in addition to reconstruction.
- Patient not suited for long surgery.
- Lack of adequate abdominal tissue.
- Patient unwilling to have additional scars on her back or abdomen.
- Small breast mound, with minimal ptosis.

*Relative contraindications*

- Young age (may need an implant replaced multiple times).
- Patient unwilling to adhere to follow-up.
- Very large/ptotic breast.
- Silicon allergy.
- Need for adjuvant radiation therapy.

*Subcutaneous implant placement*

- Visible rippling of the implant beneath a thin layer of skin.
- Risk of capsular contracture.

*Submuscular implant placement*

- Full muscle coverage, with the assistance of the serratus anterior and rectus abdominis fascia inferiorly and is, therefore, preferred.
- Coverage of the inferior pole of the implant with bioprosthetic material (e.g. human, porcine, bovine dermal allografts).
- Sutured to the pectoralis major muscle superiorly and then inferiorly to the previously marked or designated inframammary fold.

**Latissimus Dorsi reconstruction**

- Based on thoracodorsal artery.

*Indications*

- Small breast with minimal ptosis.
- Abdominal donor site unavailable (e.g. scars, lack of tissue).
- Salvage of previous breast reconstruction.

*Relative contraindications*

- Planned postoperative radiation therapy.
- Bilateral reconstruction.
- Significant breast ptosis/very large breast.
- Previous lateral thoracotomy.

**Transverse rectus abdominis muscle flap reconstruction***Indications*

- Breasts of all sizes
- Breast ptosis.

*Contraindications*

- Smoking
- Abdominal liposuction
- Previous abdominal surgery
- Pulmonary disease
- Obesity
- Patient unable to tolerate a longer procedure or a 4 to 6 weeks recovery period.

**Nipple-areolar Reconstruction**

*Done some months after the initial mound reconstruction*

- Settling of the reconstruction.
- Symmetrical positioning of the created nipple.
- A period of time after radiation should be allotted.
- Two to three months after creation of the breast mound or completion of adjuvant therapy.

*The nipple*

- Local flap techniques using the skin of the reconstructed breast mound.
- Star flap, skate flaps used.

*The areolar reconstruction*

- A full-thickness skin graft, usually from the groin for the native darker pigmentation.
- Medical tattoo pigments.

*4 to 6 weeks after creation of the nipple*

- The reconstructed nipple and areola have little projection compared with normal.
- It is insensate.

## THYROID

**Q16. Write a note on embryology of thyroid and parathyroid glands.**

**Ans.** Both the thyroid and parathyroid are derivatives of endodermal pharyngeal pouches.

**Thyroid**

- The thyroid gland develops from the **thyroglossal duct**, mainly from the 4th pharyngeal pouch. The parafollicular cells of thyroid arise from the 5th pharyngeal pouch also known as the ultimobranchial body.
- The thyroglossal duct develops as a diverticulum in the floor of the pharynx at foramen cecum just behind the tuberculum impar (a midline swelling in between the two mandibular arches) at base of the tongue.
- It then bifurcates and both leaflets of bifurcation proliferate and give rise to two lobes of the thyroid gland.

**Anomalies**

- Failure of descent from base of tongue leads to formation of lingual thyroid, intralingual thyroid, suprahyoid or infrahyoid thyroid.
- Excessive descent results in formation of intrathoracic thyroid.  
Both these anomalies of descent may be associated with a normal thyroid or may be the sole thyroid tissue present.
- Ectopic thyroid tissue may be found in trachea, esophagus, pleura, pericardium and ovary. Mass in relation to deep cervical lymph nodes are metastatic lateral aberrant thyroids and should not be confused with ectopic thyroid tissue.

- Remnants of thyroglossal duct can result in formation of thyroglossal cyst, infection in which results in thyroglossal fistula or the rare possibility of cancer.

### Parathyroid

- The superior parathyroid glands develop from the 4th pharyngeal pouch along with thyroid and is relatively constant in position
- The inferior parathyroid glands arise from the 3rd pharyngeal pouch along with thymus and travel below the superior parathyroid glands along with thymus to assume their normal position. They, however, vary in position amongst individuals and can be located anywhere from their origin near the common carotid bifurcation to neck to anterior mediastinum.

### Q17. Write a note on thyroglossal cyst and fistula.

#### Ans.

- Write embryology as in answer of Q16.
- Thyroglossal cyst is a cystic swelling in the thyroglossal duct remnant. Infection in thyroglossal cyst results in rupture and thyroglossal fistula formation.
- Thus, thyroglossal cyst is congenital whereas thyroglossal fistula is acquired.
- It presents in second or third decade of life as a midline cystic swelling in the neck.
- It is more common in women.

#### Site

- Subhyoid is the most common site.
- Other sites include suprahyoid, in the region of floor of mouth, thyroid cartilage, cricoid cartilage or at foramen cecum.

#### Clinical features

- Midline, soft cystic, fluctuant swelling in the neck that **moves up with deglutition as well as with protrusion of tongue.**
- The surface of the swelling is smooth and it is mobile sideways and not fixed to surrounding structures as well as overlying skin.
- If it gets ruptured, the opening of the thyroglossal fistula also presents at the site of the thyroglossal cyst with a **hood of skin above the opening.**
- Other complication is carcinomatous change—papillary cancer. Medullary cancer never arises in the thyroglossal cyst.

#### Investigations

- Thyroid function test.
- Thyroid scan to make sure that it is not the only thyroid tissue in the body.
- Intraoperative fistulogram is helpful in delineation of the entire tract.

#### Treatment

- **Sistrunk operation** is the surgery of choice.
- Components of surgery
  - Complete excision of the cyst or fistula.
  - Complete excision of the remnants of the thyroglossal tract.

- Excision of the central portion of the hyoid bone.
- Cosmetic skin incision and closure.

**Q18. Write a note on classification of thyroid swellings.**

**Ans.** Thyroid enlargement is called goiter.

**Its classification is as follows:**

Euthyroid simple goiter	Diffuse goiter	It is called endemic goiter when it affects more than 5% population of an area
	Multinodular goiter	
Neoplastic goiter	Benign	Colloid nodule (mc solitary nodule) Cyst Follicular adenoma
	Malignant	<b>Differentiated:</b> Papillary, follicular and medullary <b>Undifferentiated:</b> Anaplastic <b>Mesenchymal:</b> Lymphoma/ sarcoma/ metastasis
Toxic goiter	Diffuse toxic goiter	Grave's disease
	Multinodular goiter	
	Toxic solitary adenoma	Plummer disease
Inflammatory goiter	Infective	Acute (Bacterial/suppurative) Subacute (DeQuarvain/viral/granulomatous/giant cell) Chronic (tuberculosis/ syphilis)
	Autoimmune	Chronic lymphocytic thyroiditis (Hashimoto thyroiditis) Amyloidoses
	Fibrosing	Reidel's thyroiditis

**ALWAYS REMEMBER:** Important clinical features to be mentioned in any question on thyroid malignancy, thyroid nodules or goiters are as follows:

**Features suggestive of retrosternal extension**

A goiter is called retrosternal goiter if more than half of it is below the thoracic inlet.

- History of nocturnal dyspnea.
- Inability to palpate tracheal rings.
- Dilated veins in neck, puffiness and flushing of face.
- Dull percussion noted on manubrium.
- Tracheal deviation and scabbard trachea.
- Pemberton sign positive—facial congestion and respiratory distress on holding the upper limb above head in full abduction.

**Features suggestive of malignancy**

- History of dysphagia, dyspnea or hoarseness of voice.
- Hard consistency.
- Fixity to surroundings.
- Lymph node metastasis.

- Scabbard trachea.
- Horner syndrome.

**Features suggestive of metastasis**

- History of recent weight loss, anorexia, bone pains, scalp swellings
- Enlarged hard lymph nodes in neck
- Ascites/nodular liver
- Pleural effusion or consolidation

**Features suggestive of hypothyroidism**

- Hoarseness of voice.
- Dry skin.
- Decreased appetite and weight gain.
- Constipation.
- Mental lethargy, muscle weakness and fatigue.
- Cold intolerance.
- Edema of feet or moon face.
- Delayed relaxation of deep reflexes (ankle/knee).

**Features suggestive of hyperthyroidism**

- Sweating of palms and hands.
- Palpitation, exertional dyspnea, tremors.
- Eye symptoms such as diplopia, eye pain or proptosis.
- Increased appetite and weight loss.
- Diarrhea.
- Heat intolerance.
- Insomnia, menstrual disturbances.
- Tremors of involuntary muscles tested at tongue or fingers of outstretched hand.
- Tachycardia/bounding pulse evaluated using sleeping pulse rate.
- Warm and moist palms.
- Thickened skin and coarse hair.
- Bruit or thrill on the lump.

**Prognostic scores in thyroid cancer** (A- Age, M- Metastasis, E- Extrathyroid extension, S- Size, I- Invasion, G- Grade, C-Completeness of resection)

- AMES for all differentiated thyroid cancer.
- AGES for papillary thyroid cancer.
- MACIS.
- TNM.
- DeGroot and colleagues.

**Q19. Write a note on papillary thyroid cancer.**

**Ans.**

- It is the most common thyroid cancer.
- It is a differentiated thyroid cancer arising from the follicular epithelium.



**Risk factors**

- Hereditary
  - RET, MET, BRAF, RAS mutations.
  - Familial adenomatous polyposis, Werner's syndrome, McCune Albright syndrome.
- Exposure to radiation.

**Pathology**

- It is a hard tumor with multifocal lesions.
- Cut surface shows flat lesion with areas of calcification.
- Microscopy shows papillary epithelial projections, psammoma bodies and orphan annie eye nuclei.
- Spread is by lymphatics.

**Clinical features**

- Age group: 40–60 years age group.
- Sex- more common in females.
- Usually euthyroid.
- It is a painless, slowly growing, hard mass that moves with deglutition.
- Lymphadenopathy can be present.
- A lymph node in neck without thyroid swelling is a lateral aberrant thyroid which is actually a metastatic neck node of papillary thyroid cancer.

**Investigations**

- FNAC.
- Thyroid function test.
- Ultrasound neck or CECT neck.
- X-ray neck and chest.
- Indirect laryngoscopy for vocal cord mobility.

**Treatment**

- Near total or total thyroidectomy with central neck dissection for the malignancy with modified radical neck dissection on the ipsilateral side if the nodes are biopsy proven malignant.
- **Radioiodine therapy is indicated** for all the thyroid cancer patients with stage II, III, IV disease as well as for all patients with nodal metastasis, aggressive histology or vascular or extrathyroid invasion. Other indications include residual disease in neck, distant metastasis and recurrence of tumor.
- A radioiodine scan with  $I^{123}$  is done prior to radioiodine therapy. If there is significant uptake, radioiodine therapy with 30–150 mCi  $I^{131}$  is given.
- Thyroglobulin levels are used to evaluate response to the treatment as well as recurrent or metastatic disease.
- Thyroxine is given at suppression dose of 2–2.5  $\mu\text{g/kg/day}$ .
- Follow-up is done with thyroglobulin estimation 6 monthly in first year and then annually.
- Also, annual cervical ultrasound, and radioactive iodine evaluation if any of the above investigation is suspicious.

- FDG-PET scan is done when the above test results indicate recurrence and the site of recurrence is not identified.

**Q20. Write a note on follicular thyroid cancer.**

**Ans.**

- It is the second most common thyroid cancer.
- It is a differentiated thyroid cancer arising from the follicular epithelium.

**Risk factors**

- Hereditary
  - PTEN, p53, RAS mutations.
  - Carney complex type 1, Werner syndrome.
- Long standing goiter.

**Pathology**

- It is hard tumor with solitary lesion.
- Malignancy is suggested by lymphovascular and capsular invasion.
- Spread is by hematogenous route.

**Clinical features**

- Age group: 50–70 years age group.
- Sex: More common in females.
- Usually euthyroid.
- It is a painless, slowly growing, hard mass that moves with deglutition.
- Lymphadenopathy is never present.
- It can also present as pulsatile osteolytic metastasis.
- Lesions greater than 4 cm are more likely to be malignant.

**Investigations**

- FNAC is not able to differentiate between follicular adenoma and carcinoma.
- Thyroid function test.
- Ultrasound neck or CECT neck.
- X-ray neck and chest.
- Indirect laryngoscopy for vocal cord mobility.

**Treatment**

- Hemithyroidectomy with intraoperative frozen section is the procedure of choice for **FNAC showing follicular lesion.**
- If malignancy is confirmed, near total or total thyroidectomy is the procedure of choice. Lymph node dissection is not necessary as lymph node spread is not seen with this malignancy.
- Radioiodine therapy is indicated for all the thyroid cancer patients with stage II, III, IV disease as well as for all patients with nodal metastasis, aggressive histology or vascular or extrathyroid invasion.
- A radioiodine scan with  $I^{123}$  is done prior to radioiodine therapy. If there is significant uptake, radioiodine therapy with 30–150 mCi  $I^{131}$  is given.

- Thyroglobulin levels are used to evaluate response to the treatment as well as recurrent or metastatic disease.
- Thyroxine is given at suppression dose of 2–2.5 µg/kg/ day.
- Follow-up is done with thyroglobulin estimation 6 monthly in first year and then annually.
- Also, annual cervical ultrasound, and radioactive iodine evaluation if any of the above investigation is suspicious.
- FDG-PET scan is done when the above test results indicate recurrence and the site of recurrence is not identified.

**Q21. Write a note on medullary thyroid cancer.**

**Ans.** It is a differentiated thyroid cancer arising from the parafollicular C cells and secretes calcitonin.

**Risk factors**

- Hereditary
  - Isolated medullary carcinoma
  - Associated with RET mutation and multiple endocrine neoplasia syndrome II
- Sporadic medullary carcinoma with RET mutation

**Pathology**

- Most common site of origin is posterosuperiorly and laterally in the thyroid lobe.
- Amyloid in stroma is characteristic.
- It has sheets of cells
- Tumor marker is calcitonin.
- Spread is by lymphatic, hematogenous route as well as direct spread.

**Clinical features**

- Age group: It can arise in congenital form, young age if familial and 50–70 years age group if sporadic.
- Sex: More common in females.
- Usually euthyroid.
- It is a painless, slowly growing, hard mass that moves with deglutition.
- Lymphadenopathy is present.
- Patients can also have diarrhea.

**Investigations**

- FNAC.
- Thyroid function test.
- Calcium level to rule out hyperparathyroidism.
- Ultrasound neck or CECT neck.
- X-ray neck and chest.
- Indirect laryngoscopy for vocal cord mobility.
- Evaluation to rule out pheochromocytoma.

**Treatment**

- If the patient has pheochromocytoma, it is to be treated first.

- If malignancy is confirmed, near total or total thyroidectomy with bilateral central lymph node dissection with ipsilateral modified radical neck dissection irrespective of the lymph node status is the procedure of choice.
- Contralateral lymph node modified radical neck dissection is done in patients if they have even a single ipsilateral neck node biopsy proven malignant.
- If the patient has elevated calcium and/or other evidence of hyperparathyroidism, it should also be taken care of at the time of thyroid surgery.
- Radioiodine therapy is not indicated as this tumor is TSH and iodine independent.
- Calcitonin levels are used to evaluate response to the treatment as well as recurrent or metastatic disease.
- Thyroxine is given at suppression dose of 2–2.5 µg/kg/ day.
- Follow-up is done with calcitonin estimation 6 monthly in first year and then annually.
- Also, annual cervical ultrasound is done as a part of the follow-up.
- FDG-PET scan is done when the above test results indicate recurrence and the site of recurrence is not identified.

#### Prophylactic management

- Recommended for all first degree relatives and offsprings.
- Total thyroidectomy is done for all RET mutation carriers.
- In infants with MEN2A, total thyroidectomy is performed within 6 months of age and in infants with MEN2B, total thyroidectomy is performed within 12 months of age. If these infants have elevated calcitonin level, prophylactic bilateral central neck dissection is to be added.

#### Q22. Write a note on the management of a patient with solitary thyroid nodule.

Ans.

- An isolated thyroid swelling with no other clinical or radiological nodule is known as solitary thyroid nodule.
- The most common type of solitary thyroid nodule is benign colloid nodule.
- Steps of evaluation include:
  - **History and physical examination**
  - **Thyroid function test:** TSH first and if TSH shows abnormality, then proceed to T4, T3, free T4 and free T3.
    - Further evaluation depends on the thyroid function result.
    - If patient has low TSH, it suggests hyperfunction. To see if the nodule or the entire thyroid has hyperfunction, do a radioiodine scan.
  - **Low TSH and radionuclide scan result as below:**
    - If it shows **hot nodule** (hyperfunction), do surgery to resect the nodule or start patient on radioiodine treatment or antithyroid drugs. If it shows a **cold nodule**, then proceed to ultrasound to see whether the nodule is solid or cystic.
  - **Ultrasound result and further management as below:**
    - Patients with elevated TSH (hypothyroidism) or normal TSH undergo USG. Also patients with low TSH with a cold nodule on radionuclide scan should undergo ultrasound as a part of their further evaluation.

- **Cystic lesion** can be aspirated for maximum 3 times. If it then recurs or if the aspirate shows malignant cells, do surgery.
- **Solid lesion** is finally subjected to FNAC.
- **FNAC grading is given by Ishiki** and is as follows:

1	Nondiagnostic	Repeat the FNAC
2	Non-neoplastic/ Benign	Cyst managed as mentioned above. Colloid nodule managed with thyroid replacement and if it is symptomatic, compression symptoms or cosmetic reason or size > 4 cm, consider surgical resection.
3	Indeterminate/ follicular	Radioiodine scan and if cold nodule than hemithyroidectomy with frozen section. If it shows malignancy in frozen section, manage as per follicular cancer. If no malignancy, follow up. Hot nodule in scan – surgery, radioiodine or antithyroid drugs.
4	Suspicious of malignancy	Do hemithyroidectomy with frozen section. If frozen section is showing malignancy, do manage as per the type of malignancy
5	Definite malignancy	Manage as per the type of malignancy

#### Risk of malignancy in STN

- Male has more risk of malignancy than female.
- Solid has more risk of malignancy than cystic.
- Cold nodule has more risk of malignancy than hot nodule.
- Slow growing nodule has more risk of malignancy than rapidly growing nodule.
- Young patient has more risk of malignancy than old patient.
- Solitary nodule has more risk of malignancy than multinodular goiter.
- Patients with family history of thyroid malignancy or radiation exposure have more risk of malignancy.
- Risk in solid STN in males is 50–40% and in cystic STN is 30–20%. Risk in solid STN in females is 20–10%, whereas in cystic STN is 10–5%. (Notice the sequence to remember the values.)

#### Q23. Write a note on Grave's disease.

##### Write a note on management of diffuse toxic goiter.

**Ans.** It is the most common cause of primary hyperthyroidism.

##### Pathophysiology

- Formation of autoantibodies to TSH receptor which persistently stimulate the receptor and produce hyperthyroidism.
- HLA B8, DR3, DQA1, CTLA4 predispose, whereas HLA DR B1 0701 is protective.
- It is also associated with other autoimmune conditions such as type 1 diabetes, Addison's disease, myasthenia and pernicious anemia.

##### Clinical features

- Signs of hyperthyroidism.

- Diffuse goiter.
- Pretibial myxedema with hyperpigmented skin and thyroid acropachy.
- **Eye signs**
  - Lid retraction (earliest sign).
  - Lid lag (Von Graefe sign).
  - Dalrymple sign (staring look).
  - Stellwag sign (infrequent blinking).
  - Moebius sign (convergence defect).
  - Joffroy sign (absence of wrinkles on forehead on looking up).
  - Exophthalmos (Naffziger method to look for it).
  - Gifford sign (difficulty to evert eyelid).
  - Involvement of superior and lateral rectus and inferior oblique leads to inability to look upward and outward.
  - Corneal ulcers, conjunctival chemosis and diminished vision in later stages.
- Patient can also have gynecomastia and milky nipple discharge.

### Investigations

- Thyroid function tests.
- Thyroid autoantibodies test to TSH-R.
- Radioiodine scans show increased uptake.
- MRI of eye for the extent of ophthalmopathy.

### Treatment options and selection

<b>Surgery</b>	<p><b>Indications</b></p> <ul style="list-style-type: none"> <li>• Failure of medical or radioiodine therapy</li> <li>• Risk of malignancy cannot be ruled out</li> <li>• FNAC shows indeterminate or suspicious result</li> <li>• Compressive symptoms</li> <li>• Cosmetic reason</li> <li>• Noncompliance or complications to medications</li> <li>• Contraindications to radioactive iodine therapy</li> <li>• Smokers with Grave's ophthalmopathy do better with surgery</li> </ul> <p><b>Patients should be given Lugol's iodine</b> therapy for 5–10 days preoperatively as it decreases the size and vascularity of thyroid gland</p> <p><b>Antithyroid drugs</b> given to make patient euthyroid before surgery</p>
<b>Antithyroid medications</b>	<ul style="list-style-type: none"> <li>• Propylthiouracil is the drug of choice</li> </ul> <p><b>Mechanism</b></p> <ul style="list-style-type: none"> <li>• Inhibit iodide coupling</li> <li>• Inhibits peripheral conversion of T4 to T3</li> <li>• The symptom resolution requires 6–8 weeks</li> <li>• Usually used only to make patients euthyroid before radioiodine therapy or surgery</li> <li>• As solo therapy, they are of help only in the patients with small goitres and mild hyperfunction of thyroid</li> </ul> <p><b>Side effects</b></p> <ul style="list-style-type: none"> <li>• Peripheral neuritis, polyarteritis, agranulocytosis, vasculitis, aplastic anemia.</li> <li>• Methimazole is associated with aplasia cutis, therefore, not used in pregnancy</li> </ul>

<b>Radioiodine therapy</b> $I^{131}$ is used. It emits beta rays (90%) and gamma rays (10%)	<b>Indications</b> <ul style="list-style-type: none"> <li>• Older patients in whom surgery or antithyroid medications are contraindicated</li> <li>• Relapse after medical or surgical therapy</li> <li>• Patient's preference of not requiring surgery</li> </ul> <b>Advantages</b> <ul style="list-style-type: none"> <li>• No surgery</li> <li>• Ease of procedure and cheap</li> </ul> <b>Disadvantages</b> <ul style="list-style-type: none"> <li>• Increase in cardiac mortality</li> <li>• Increase in risk of thyroid cancer</li> <li>• Progression of Grave's ophthalmopathy</li> <li>• Contraindicated in pregnancy, lactation and patients with ophthalmopathy</li> </ul>
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**Q24. Write a note on complications of thyroidectomy.**

**Ans.**

- **Hemorrhage**
  - **Primary hemorrhage:** Hemorrhage due to inadequate hemostasis.
  - **Reactionary hemorrhage:** Due to slippage of ligature or clot on normalization of blood pressure on coming out of anesthesia or drug effects.
  - **Secondary hemorrhage:** Due to infection or necrosis or malignancy eroding into vessel and causing hemorrhage.
- **Parathyroid insufficiency**
  - Cause is vascular deprivation of parathyroid more often than inadvertent removal of parathyroid.
  - Mild hypocalcemia is taken care of by oral supplements and milk.
  - Severe hypocalcemia (<7 mg/dL) or symptomatic hypocalcemia (tingling, numbness, Chvostek's sign, Trousseau sign) indicates parenteral calcium gluconate IV to be given under ECG guidance and repeated if symptoms do not resolve.
  - Once symptomatic relief is obtained. Start patient on oral calcium with Vitamin D<sub>3</sub> with total dose even up to 2–2.5g/day in 4–6 divided doses.
- **Injury to recurrent laryngeal nerve**
  - It can present in immediate postoperative period as dyspnea or hoarseness of voice or stridor.
- **Injury to external branch of recurrent laryngeal nerve**
- **Injury to cervical sympathetic chain**
  - Present as Horner syndrome—ptosis, miosis, anhidrosis, enophthalmos, loss of ciliospinal reflex, heterochromia iridis.
- **Suture hematoma**
- **Laryngeal edema**
- **Hypothyroidism**
- **Thyroid storm**
  - If a hyperthyroid patient is not properly prepared preoperatively.
- **Wound infection or abscess**

## PARATHYROID AND ADRENAL

### Q25. Write a note on primary hyperparathyroidism.

**Ans.** Primary hyperparathyroidism is increased production of parathyroid hormone from parathyroid glands.

#### Pathophysiology

- Parathyroid adenoma is the most common cause.
- Amongst parathyroid hyperplasia, chief cell hyperplasia is the most common cause. Parathyroid hyperplasia is the term used when three or more glands are involved by the disease.
- Etiology leading to parathyroid hyperfunction can be renal dysfunction leading to hypocalcemia and constant parathyroid stimulation. Familial cause such as MEN1 syndrome or MEN2A syndrome wherein parathyroid involvement and hypercalcemia is the most common and most early manifestation, exposure to radiation.
- It is more common in postmenopausal females.

#### Clinical features

The traditional symptom line of hyperparathyroidism is as follows:

- Bones, moans, groans, stones and fatigue overtones.
- Detailed as bone pain, psychiatric symptoms such as depression, memory loss, abdominal colics (groans), kidney stones, and muscle weakness and fatigue.
- Other common symptoms of hypercalcemia include polyuria, nocturia, polydipsia and constipation.

#### Diagnoses

- Elevated serum PTH.
- Elevated serum calcium.
- Decreased serum phosphate.
- Normal urinary calcium.

#### Imaging localization of disease

- **X-rays of hands** show osteitis fibrosa cystica or Von Recklinghausen disease of bone. It is characterized by bone cysts, tufting of middle phalanges and subperiosteal bone resorption especially in middle phalanges.
- **Ultrasound neck + sestamibi scan** when used together and when both give same result, it is 95% accurate.
- **Sestamibi (technetium)** is the most accurate imaging investigation in preoperative phase. It can also be used intraoperatively in radio-guided parathyroidectomy.
- **Ultrasound neck** is mainly used for juxtathyroid or intrathyroid parathyroids.
- **CECT neck and chest** is done to rule out intrathoracic parathyroids.
- Intraoperatively, greater than 50% decrease in parathyroid level 10 minutes after the excision of the said pathology confirms treatment.

#### Treatment

- Correction of fluid and calcium abnormality followed by surgery.



**Indications of surgery**

- Age <50 years.
- Older age patients with psychiatric symptoms.
- Life threatening episode of hypercalcemia.
- Presence of renal stones.
- Patient preference.
- Serum calcium elevated >1 mg/dL greater than the higher limit of normal.
- Elevated 24 hour urinary calcium level (>400 mg/day).
- Creatinine clearance reduced by 30%.
- Decreased bone mineral density.
  - During surgery, resection is the procedure of choice. Remove all the diseased parathyroids.
  - If all 4 are diseased, then autoimplantation of 1/2 of 1 gland is necessary and can be done in sternomastoid or brachioradialis muscle.
  - If none of the four is diseased, then search should be made for ectopic or supernumerary parathyroid glands in paraesophageal (mc site) and mediastinal locations. Sternotomy, “walking down” the thymus, cutting thyrothymic ligament, and exploring the thyroid are all measures to search for the parathyroids.

**ALWAYS REMEMBER:** Indications of surgery in secondary hyperparathyroidism are as follows:

- Pathological fractures.
- Bone pains.
- Ectopic calcifications.
- Intractable pruritus.
- Open ulcerative skin lesions due to calcinosis.

**Q26. Outline the management of adrenal incidentaloma.**

**Ans.** Incidentaloma is an adrenal lesion that is incidentally discovered on an investigation done for some other purpose.

It can be functioning or nonfunctioning.

**The management outline is as follows:**

**History:** If the patient was being worked up for malignancy or if the lesion is bilateral or if the patient has a history of malignancy, then the incidentaloma is likely to be metastasis.

**Evaluation for function**

<b>Pheochromocytoma</b>	Plasma metanephrine is the best test Other tests include urine 24 hour metanephrine Urine VMA Biopsy is contraindicated
<b>Aldosteronoma</b>	Plasma aldosterone/plasma renin ratio is the best test
<b>Cushing or adrenocortical carcinoma</b>	Low dose or high dose dexamethasone suppression test Serum cortisol level in early morning

- If it is functioning tumor, proceed straight to excision.
- If it is non-functioning tumor, proceed to radiological investigation dedicated for adrenals as described in the radiology section.

**Indications of surgery in incidentaloma:**

- Functioning tumor.
- Metastasis.
- Primary malignancy.
- All lesions >5 cm.
- Tumors 3–5 cm with rapid growth in the observation period/marked heterogeneity/irregular margins/patient preference/young age.

**Laparoscopy can be performed for localized tumors <5 cm and manageable lymphadenopathy.**

- **If it turns out to be pheochromocytoma**, then do a dedicated whole body MRI to rule out other associated paragangliomas (10% incidence, the most common site is organ of Zuckerkandl at bifurcation of aorta to left of origin of inferior mesenteric artery).  
**(ALWAYS REMEMBER: Zuckerkandl tubercle** is lateral and posterior most part of thyroid that points towards the recurrent laryngeal nerve. **Zuckerkandl fascia** is posterior layer of renal fascia and **Zuckerkandl organ** is the most common site of paraganglioma.)
- Do adequate fluid management of patient.
- Start him on alpha-blockers first, and once the alpha blockade is adequate, start on beta-blockers if needed to control heart rate and have a cardioprotective effect.  
**(ALWAYS REMEMBER: Beta comes after alpha.)**
- Surgery is done only after these factors have been taken care of.
- Also evaluate the patient for presence of MEN syndromes, VHL, neurofibromatosis, Sturge-Weber syndrome, tuberous sclerosis before surgery.

# SECTION

# 7

## Surgical Radiology



**Q1. Discuss the basic principles of computed tomography (CT).**

**Describe in brief the CT principles with advantages and disadvantages of the modality.**

**Ans.** Computed tomography (CT) is X-ray based imaging modality that uses computer processed X-rays to produce tomographic images (virtual slices) of specified area of the scanned object, allowing the user to see what is inside without cutting it open. The first commercially-used CT machine was invented by Sir Godfrey Hounsfield in 1973.

**Limitation of X-rays**

Conventional X-ray imaging is two-dimensional projection image of a three-dimensional structure, leading to overlap of structures in resultant image with difficulty in interpretation of complex anatomical structures like skull and pelvis. This was the major limitation of conventional X-ray imaging.

The **basic principle of tomography** is to rotate the X-ray source and detector array around the plane of object of interest, keeping the object at the focal point of rotation, so that structures out of plane of interest appear blurred due to motion artifact, reducing the effect of overlapping. The conventional tomography is currently widely used in dental radiology for orthopantomogram (OPG).

**CT is different from conventional tomography in following manner:**

- CT uses the X-ray source producing fan-shaped beam of X-rays on one side of the patient and detector array on the opposite side, with both rotating simultaneously around the patient in opposite direction in a technologically-advanced support structure called CT gantry.
- The resultant projection of X-rays through particular section of the body, is detected by the opposite side detector array and their attenuation by body section is calculated by complex computer-assisted mathematical algorithms, so called computed tomography (CT). The thickness of the section of the body scanned by one gantry rotation is determined by width of X-ray beam and detector array, increasing either will lead to increase in section thickness and scan speed at the cost of spatial resolution.
- After one gantry rotation, patient table move along Z axis (long axis of the patient), and gantry rotates again producing cross-sectional image of the next slice of the patient. This process continues till the volume of interest is scanned completely.

**How CT image generated from projection of the X-rays?**

- The slice of the patient is divided into multiple small unit cubes, called voxels. Each voxel contain different tissue or combination of tissues, which attenuate X-rays in various proportions.
- Average attenuation of X-rays by tissue in particular voxel is determined by linear attenuation coefficient of that voxel.
- Depending on the linear attenuation coefficient, each voxel is assigned a CT number or Hounsfield number or unit (HU value) by following formula:

**HU value or CT number = attenuation coefficient of voxel – that of water/ that of water**

- Depending on the HU value, each voxel is assigned a particular gray scale value (that depends on bit depth of analogue to digital converter and window setting), that will be seen as corresponding pixel in resulting cross-sectional image.
- From the formula, it is apparent that water has zero HU value.
- Fat has HU value in range of  $-10$  to  $-50$  or  $-100$  (it attenuated X-rays less than water) appear hypodense (dark) on image.
- Air has much more negative HU value in range of  $100$ s, appearing black on resultant image.
- Bone, contrast material, calcification, etc. attenuate X-rays more than water, having higher HU values and appear hyperdense (bright) on resultant image.

### **Advances in CT technology**

- Nowadays with advancement of the X-ray tube having better heat loading capacity and compact structure, gantry rotation time is greatly reduced, leading to faster scans. Further advancement in gantry design, slip ring technology, fast post processing of the data and better computational powers lead to invention of helical CT, where gantry rotates continuously and simultaneously patient moves along Z axis, providing helical or spiral sections of the patient.
- Multidetector or multislice CT (MDCT) utilizes multiple rows of detectors along Z axis of the patient (6, 16, 64, 128, 256, 316, etc. number describes the number of rows of detectors along Z axis of the patient), with the significant advantage of high speed scanning and isotropic resolution.
- Dual energy CT uses two X-ray tubes in the same gantry, operating at different KV (80 and 140 KV most commonly). The basic principle of dual energy CT is different attenuation of X-rays by different materials at two different KV, depending on their atomic number due to differential photoelectric effect, allowing material decomposition of the lesion. It helps in detection of composition of the renal stone, gout crystals, atherosclerotic plaques, etc.
- Cone beam CT uses cone-shaped X-ray beam instead of fan-shaped beam, producing images within single gantry rotation with high-speed imaging. It is commonly used in dental radiology as office-based compact CT scanner. Cone beam CT can also be mounted on mobile C arm equipment for use in orthopedic operation theaters and interventional radiology.
- Electron beam CT is further advancement, commonly used for cardiac and coronary CT.

### **Advantages**

- Cross-sectional imaging modality, providing images without overlap.
- Rapid imaging modality compared to MRI, helpful in imaging of lungs and abdomen as very short duration of breath hold is required. Pediatric CT or patients with altered sensorium and uncooperative patients can be scanned with short-term sedation with midazolam, while magnetic resonance imaging (MRI) requires longer duration of sedation with need of general anesthesia for the same purpose.
- Excellent for the detection of fat, air, bone and calcification. On MRI air, bone and calcification appear black or spin echo sequences due to lack of protons in all of them, while CT can differentiate them having different attenuation coefficients.
- Various advancements lead to newer applications of CT, including CT angiography, CT urography, CT myelography, perfusion CT, etc. that have eliminated the need of various interventional procedures.

- CT-guided interventions, particularly for those structures which are not accessible or difficult to see on ultrasound are very helpful, e.g. CT-guided lung biopsy, biopsy from spine.
- It has excellent spatial resolution, better than MRI.

**Limitations**

- It uses X-rays, giving ionizing radiation to the patient, the major issue with the CT scan. It has very limited use in pregnant patients.
- Soft tissue resolution is poor as compared to MRI, hence requires iodinated contrast injection most of the time for better tissue differentiation.
- Iodinated contrast agents used for CT scanning have their own risk factors and side effects, including anaphylactic and allergic reactions, contrast-induced nephropathy, etc.
- Because of beam hardening artifacts, CT has limited capacity to demonstrate soft tissue surrounded by bony structures like posterior fossa of skull, pelvis, spinal cord, etc. MRI provides excellent soft tissue contrast for the similar structures.
- Availability and cost may be the limiting factors in developing countries.

**Q2. Discuss basic principles of ultrasound.**

**Write short note on ultrasound with its advantages, limitations and basic applications.**

**Ans.**

- Ultrasound is an oscillating pressure sound wave with a frequency (3 to 20 MHz) greater than the human audible range.
- Medical or diagnostic ultrasonography uses ultrasound produced by the transducer to image various organs and parts.

**What is piezoelectricity?**

- The basic principle of sonography is piezoelectricity or piezoelectric property.
- Piezoelectricity refers to a characteristic property of certain elements, which on application of electronic energy vibrate and convert it into mechanical energy (ultrasound) and vice versa.
- The most commonly utilized piezoelectric element for sonography is compressed microcrystalline lead titanate zirconate (PZT). Quartz also has piezoelectric property, but commonly utilized in digital timers and computers.
- These microcrystalline PZT is arranged in highly organized manner on a plastic hand held device, called ultrasound transducer or probe. Depending on the geometry and structural properties of PZT, transducer produce different range of sound frequencies, which is used for imaging of various organs located at different depth from the skin surface.

**From sound to image**

Formation of image from sound occurs in three steps.

*Producing a sound*

- Sound is produced by piezoelectric effect of transducer as described.
- Water-based gel is applied over the transducer for acoustic coupling of transducer to body and preventing attenuation of sound in intervening air.

- The sound wave undergo various fates in body like reflection, attenuation, refraction depending on the physical properties of various tissues, density of organs, angle of incident and attenuation coefficients.
- Some of the reflection returns to the transducer.
- Higher the frequency of the sound, more rapid attenuation occurs, however, it produce high resolution images, hence high frequency ultrasound transducer is used for imaging of superficial structures like testis, thyroid gland, breasts, bowel loops, pleural cavity, muscles, tendons, joint spaces etc. For scanning of various abdominal organs, located deep from the skin surface, low frequency curvilinear transducer (3 to 5 MHz) is used.

#### *Receiving the echo*

- The return of the sound wave to the transducer results in the same process that it took to send the sound wave, except in reverse.
- The return sound wave vibrates the transducer; transducer turns vibrations into electrical pulses that travel to the ultrasonic scanner where they are processed and transformed into a digital image.

#### *Forming the image*

The sonographic scanner must determine three things from each received echo:

- a. How long it took the echo to be received from when the sound was transmitted.
- b. From this the focal length for the phased array transducer is deduced, enabling sharp image of that echo at that depth.
- c. How strong the echo was.

Once the ultrasonic scanner determines all the three things, it can locate which pixel in the image to light up and to what intensity and at what hue if frequency is processed.

#### **Modes of ultrasound**

- **A-mode:** A-mode (amplitude mode) is the simplest type of ultrasound. A single transducer scans a line through the body with the echoes plotted on screen as a function of depth. Therapeutic ultrasound aimed at a specific tumor or calculus is also A-mode, to allow for pinpoint accurate focus of the destructive wave energy.  
It is commonly used for examination of eye, detection of cyst in breast and detection of midline shift in brain.
- **B-mode or 2D mode:** In B-mode (brightness mode) ultrasound, a linear array of transducers simultaneously scans a plane through the body that can be viewed as a two-dimensional image on screen. More commonly known as 2D mode now.
- **M-mode:** In M-mode (motion mode) ultrasound, pulses are emitted in quick succession—each time, either an A-mode or B-mode image is taken. Over time, this is analogous to recording a video in ultrasound. As the organ boundaries that produce reflections move relative to the probe, this can be used to determine the velocity of specific organ structures.
- **C-mode:** A C-mode image is formed in a plane normal to a B-mode image. A gate that selects data from a specific depth from an A-mode line is used; then the transducer is moved in the 2D plane to sample the entire region at this fixed depth. When the transducer traverses the area in a spiral, an area of 100 cm<sup>2</sup> can be scanned in around 10 seconds.



- **Doppler mode:** This mode makes use of the Doppler effect in measuring and visualizing blood flow:
  - **Color Doppler:** Velocity information is presented as a color-coded overlay on top of a B-mode image
  - **Continuous Doppler:** Doppler information is sampled along a line through the body, and all velocities detected at each time point are presented (on a timeline)
  - **Pulsed wave (PW) Doppler:** Doppler information is sampled from only a small sample volume (defined in 2D image), and presented on a timeline
  - **Duplex:** A common name for the simultaneous presentation of 2D and (usually) PW Doppler information (Using modern ultrasound machines, color Doppler is almost always also used; hence the alternative name **Triplex**).
- **Pulse inversion mode:** In this mode, two successive pulses with opposite sign are emitted and then subtracted from each other. This implies that any linearly responding constituent will disappear while gases with nonlinear compressibility stand out. Pulse inversion mode is commonly used with contrast-enhanced ultrasound (CEUS).
- **Harmonic mode (tissue harmonic imaging or THI):** In this mode a deep penetrating fundamental frequency is emitted into the body and a harmonic overtone is detected. This way noise and artifacts due to reverberation and aberration are greatly reduced.

### Advantages

- Ultrasound is cheap, easily available and commonly used imaging modality with widespread clinical applications.
- It does not cause ionizing radiation to patients, hence commonly used for obstetrics and gynecological imaging.
- Mobile ultrasound equipment can be used for bedside ultrasound in ICU settings.
- It can live real time imaging of various structures thus enabling various vascular and nonvascular interventions.
- It does not require injection of intravenous contrast material like CT and MRI.
- Patient motion is not a major issue, hence long-term sedation and general anesthesia can be avoided.

### Limitations

- It is an operator dependent modality, hence poor reproducibility and interobserver agreement.
- Ultrasound beam cannot penetrate through gas; hence air-containing structures cannot be visualized like lungs, bowel lumen. Patients are, therefore, advised to come empty stomach for abdomen ultrasound. The rationale of full urinary bladder for pelvis ultrasound is to displace the gas containing bowel loops superiorly, and urine containing bladder acting as ultrasound window for various pelvis organs.
- Ultrasound cannot penetrate through cortical bone, hence it has limited role in evaluation of intraosseous, intraspinal and intracranial pathologies.
- Patient cooperation is required in terms of breath holding.
- Soft tissue resolution is poor as compared to MRI.
- Spatial resolution is poor as compared to CT.

**Applications**

- It is the imaging modality of choice for various hepatobiliary indications including liver abscess, gallstones, cholecystitis, etc.
- Primary imaging modality for renal stones and hydronephrosis detection.
- Ultrasound is most commonly used imaging modality for diagnosis of acute appendicitis.
- Ultrasound is primary imaging modality for various benign and malignant conditions of female pelvis, e.g. uterine fibroid, ovarian cysts, ovarian carcinomas, hydrosalpinx, etc.
- Ultrasound is also used for assessment of prostate size and zonal anatomy.
- It has widespread applications in obstetrics right from the confirmation of diagnosis of pregnancy, fetal monitoring, growth assessment, detection of various congenital anomalies, etc.
- Doppler ultrasound is used for assessment of various vascular pathologies, like peripheral vascular disease, varicose veins, deep venous thrombosis, portal hypertension, renal artery stenosis, and atherosclerotic involvement of carotid arteries.
- Neonate and infants have open fontanelle, allowing ultrasound assessment of various neurological conditions.
- Ultrasound also helps in various interventions like liver abscess drainage, ascetic and pleural effusion tap, TRUS-guided prostate biopsy, renal biopsy.

**Advances in ultrasound**

- Different designs of ultrasound transducers are available for special purposes. Endocavitary probes for transvaginal and transrectal ultrasound allow use of high frequency as distance between probe and target is reduced.
- 3D and 4D ultrasound is helpful in detail anatomical evaluation of complex anomalies and pathologies having primary role in obstetrics and gynecology.
- Contrast-enhanced ultrasound (CEUS): Various ultrasound contrast agents (sonovue available in India) are microbubbles of gas (most commonly sulfur hexafluoride) with sugar, protein or lipid as stabilizing shell. They allow evaluation of hemodynamic properties like CT and MRI contrast agents.
- Elastography: Ultrasound elastography is a newer imaging technique that detects stiffness or elasticity of the tissue by measuring the velocity of soundwaves. Pathological tissues and fibrosis have higher stiffness and higher velocities, allowing noninvasive detection of liver fibrosis, cirrhosis and various malignancies.
- Therapeutic ultrasound: In addition to diagnostic uses, ultrasound also has some therapeutic applications. HIFU (high intensity focused ultrasound) uses well-concentrated high frequency ultrasound beam to generate heat and ablate the pathological tissue.

**Q3. Describe basic principles of Doppler ultrasound and role of Doppler in assessment of peripheral vascular diseases.**

**Ans.** The Doppler Effect is familiar to those heard the siren sound on an emergency vehicle as it passes by.

**Doppler effect**

- When incident sound waves  $I$  of frequency  $f$  are reflected at right angles by a moving interface that is approaching a transducer, the waves are compressed. The wavelength

is reduced and the frequency  $f$  of reflected sound wave is increased. With the receding reflector, the frequency is reduced.

- The change of frequency is proportional to the velocity of the interface.
- The higher the transducer frequency or interface velocity, higher the Doppler frequency shift.
- The change of the frequency is measured and shows how fast the reflector is moving and direction of movement whether towards or away from the transducer.
- The resultant frequency shift is depicted as color coding in the image. Red color describes increase in frequency and the interface movement towards the transducer and blue color indicates decrease in frequency with movement of the interface away from the transducer; however, this color setting can be inverted manually on the machine.
- Various mode of Doppler ultrasound are described in previous question.
- Commonly used at present is duplex ultrasound, that is gray scale B-mode ultrasound image showing color Doppler findings on it and spectral waveform can also be calculated from small area of interest from the various vascular structures.

### **Radiological evaluation of peripheral vascular disease (PVD)**

#### *Normal arterial waveform*

- The normal spectral Doppler pattern of extremity arteries is typical high resistance waveform. It consists of a narrow sharply defined tracing indicating that all blood cells are moving at an equivalent speed at any time in cardiac cycle. The configuration of this waveform is typically triphasic, indicating strong forward component of flow during systole, followed by a short reversal of flow during diastole.
- A return to forward flow of lower amplitude normally follows and lasts for a variable length of diastole.
- The diastolic component of the blood flow is extremely variable disappearing with the vasoconstriction due to cold or increasing with warmth or exercise. The diastolic component is absent in vessels that have lost compliance as in atherosclerotic and diabetes.

*The radiological evaluation of PVD is aimed at providing principally following information:*

- Presence of any hemodynamically significant obstruction to the vascular flow.
- Exact site and extent of the block.
- Status of collateral flow and distal vasculature for management planning.
- Evaluation of results of therapy and disease progression.

Doppler imaging provides several precious clues that can be used to detect significant obstruction. Color aliasing, persistence (forward continuous flow) and color bruit (periarterial vibrations) are helpful indicators of the flow disturbances with significant stenosis.

Once the abnormal flow pattern is detected by color Doppler, pulsed Doppler spectral sampling is done to better characterize the lesion.

Focal peripheral vascular stenosis is categorized as mild, moderate or severe:

- With the mild stenosis (1–19% luminal diameter reduction), pulse Doppler reveals normal triphasic waveforms with mild spectral broadening. PSV is up to 30% greater than the velocity of the normal proximal segment.

- With the moderate stenosis (20–49% reduction of luminal diameter), the waveform remains triphasic but further spectral broadening occurs. PSV may increase up to 100% greater than the proximally sampled segment.
- A severe stenosis represents a 50 percent or greater reduction in luminal diameter. These lesions are considered hemodynamically significant as it results in decreased blood pressure and blood flow across the stenosis. There is marked spectral broadening with spectral waveform distal to the stenosis is monophasic indicating reduced vascular resistance. PSV at the level of stenosis is more than double the proximal segment PSV.
- PSV ratio greater than 4 indicates more than 80% and ratio more than 7 indicates more than 90% stenosis. An occlusion is characterized by absence of flow within an arterial segment.

A PSV ratio is more relevant than absolute PSV values, as ratio is independent of angle of ultrasound beam incident and various other hemodynamic variations.

Distal to the level of critical stenosis, the waveform shows a typical low resistance pattern due to opening up of collaterals and loss of normal arterial tone. There is also a characteristic tardus-parvus waveform in the distal vessel.

#### **Q4. Discuss basic principles of magnetic resonance imaging (MRI).**

**Describe principles of MRI in brief with advantages and disadvantages of the modality.**

**Enumerate the contraindications of MRI.**

**Ans.** Four basic steps are involved in getting an MR image:

- a. Placing the patient in MR magnet
- b. Sending the radiofrequency pulse by transmitter coil
- c. Receiving signal back from the patient by receiver coil
- d. Received signals are sent to computers for complex processing by Fourier transmission for final image reconstruction.

Present MR imaging is based on proton imaging. Proton is a positively charged particle in the nucleus of every atom. Since hydrogen ( $H^+$ ) has only one particle, i.e. proton, it is equivalent to a proton. Theoretically, any atom having odd number of protons or charged particles generate net magnetization around them when placed in external magnetic field, hence can be used for MR imaging, e.g.  $^{13}C$ ,  $^{19}F$ ,  $^{23}Na$ ,  $^{31}P$ . Hydrogen ions are present in abundance in body water.  $H^+$  gives best and most intense signal among all nuclei.

**How does this ions (protons) help in MR imaging?**

- Protons are positively charged and have rotatory movement called spin. Any charge which moves, generates current. Every current has a small magnetic field around it. So every rotating proton has a small magnetic field around it.
- In absence of any external magnetic field, protons move in random direction in body. In the presence of external magnetic field, these randomly moving protons align themselves in the direction of external field ( $B_0$ ), with some of them aligning parallel and others in antiparallel direction.
- When protons align, not only they rotate but their axis of rotation also moves in such a manner that it forms a cone. This movement of an axis of rotation is called precession. The number of precession of protons per second is called precession frequency in Hertz.

Precessional frequency is directly proportional to the strength of external magnetic field. This relationship is expressed by Larmor's equation

$$W_0 = \gamma \cdot B_0$$

Where,  $W_0$  = precessional frequency in Hz

$B_0$  = Strength of external magnetic field

$\gamma$  = gyromagnetic ratio, which is specific to particular nucleus

### Magnetization

- For the orientation in space consider X, Y and Z axis. External field is applied along Z axis. Conventionally, Z axis is along the long axis of the patient as well as bore of magnet. Protons align parallel and antiparallel to Z axis. Forces of protons on positive and negative Z axis cancel each other. However, there are always more protons spinning on positive side or parallel to Z axis than negative side, so after cancelling each other, few protons remain on positive side, which are not cancelled. Forces of these protons add up together to form a magnetic vector along Z axis called **longitudinal magnetization**.
- Longitudinal magnetization along the direction of external magnetic field cannot be measured directly. For measurement, it has to be transverse.
- As the radiofrequency pulse is sent, the precessing protons pick up some energy from radiofrequency pulse. Some of the protons go to higher energy stage and start precessing in anti-parallel direction. This results in reduction in longitudinal magnetization. Forces now add up to generate new magnetic vector in transverse plane (XY plane), called **transverse magnetization**.
- For exchange of energy to occur between protons and RF pulse, precession frequency of protons should be same as RF pulse energy.

### MR signal

- Once the radiofrequency pulse is removed, the nuclei realign themselves with the main magnetic field (relaxation) and in the process emit a radiofrequency signal that can be recorded and spatially encoded. MR signal is Fourier transformed into MR image by computers.
- The specific tissue characteristics define the manner and the rate at which these nuclei relax. This relaxation is measured in two ways, referred to as T1 and T2 relaxation times. These relaxation times and proton density determine the signal from a specific tissue.
- There are large number of imaging sequences that can be used by applying radiofrequency pulses of different strength and durations. The image characteristic and signal intensity from different tissues are governed by the pulse sequence employed and whether it is T1 weighted or T2 weighted. For instance, fat, methHb and mucinous fluid appear bright on T1WI, water and thus most of pathological processes which tend to increase tissue water content appear bright on T2WI. Cortical bone, air, hemosiderin and ferromagnetic materials are of very low signal on all pulse sequences.
- In general, T1WI are better for anatomical delineation and T2WI are better for pathological processes. For added, tissue contrast, intravenous gadolinium can also be injected followed by fat saturated T1WI. Nowadays, other more specific contrast medium for imaging of liver, nerves and lymph nodes are available.

### Advantages of MRI

- No ionizing radiation.

- *Excellent soft tissue contrast:* MRI provides images with exquisite soft tissue contrast resolution, enabling differentiation of various soft tissues, which was not possible with CT and ultrasound.
- MRI has also the advantage of multiplanar imaging, as images can be acquired in any direction and plane.
- It has traditionally been used extensively in assessment of intracranial lesions, spinal lesions and various musculoskeletal disorders.
- More recent developments have resulted in new indications and applications.
- Nowadays, MRI is commonly used in oncology imaging, such as staging of various carcinomas, breast malignancy, characterization of various hepatic masses, assessment of biliary tree (by MRCP).
- Various pelvic disorders and malignancies are better evaluated with MRI due to lack of bone hardening artifacts seen in CT.
- MR angiography can be carried out without intravenous injection of contrast in completely non-invasive manner by newer techniques [like time of flight (TOF) and phase contrast (PC) MRI].

#### *Limitations*

- The availability of MRI is limited at many places.
- It is time consuming as compared to other cross-sectional imaging modalities.
- Images are easily degraded by motion of patient, including respiratory and cardiac motion. The use of respiratory and cardiac gating can eliminate this, although bowel peristalsis can still be problem. The long acquisition time require very cooperative patient who can lie still for long enough time to be scanned which is difficult for claustrophobic patients or those who are in pain.
- Long-term sedation or general anesthesia may be required for uncooperative or pediatric patients.
- It is expensive imaging modality compared to others.

#### **Contraindications**

There are several absolute contraindications for MR imaging.

#### *Patient with aneurysm and hemostatic clips*

- Many of these clips are ferromagnetic are absolute contraindication to MRI imaging. Only those clips which are made of titanium and documented by manufacturer are allowed to enter into the magnetic field.

#### *Ocular implants*

- There is risk of discomfort and minor injury with ocular implants.

**Ontological implants:** Cochlear implants are absolute contraindication to undergo MR imaging.

#### *Pellets, bullets and sharpnells*

- Patients with bullet injury, shrapnel within body or pellets are contraindicated for MR imaging.

#### *Penile implants and artificial sphincters*

**Cardiac pacemakers**

- There is possibility of displacement and damage of pacemaker, programming change, electromagnetic interference and fibrillation when patient with cardiac pacemaker undergo MR examination.

**Implantable cardiac defibrillators***Vascular access ports*

- Ports with electronic activation and programming are strict contraindication.

**Neurostimulators****Bone growth stimulators****Intraocular metallic foreign bodies**

**Relative contraindications** are first trimester pregnancy and claustrophobia.

Patients with dental devices and materials, heart valves, intravascular coils, stents and filters, orthopedic implants are no longer considered contraindicated for MR imaging.

**Q5. Discuss in brief the principle of PET scan and the role of PET scan in diagnostic radiology.**

**Write a short note on PET scan.****Ans.**

- Positron emission tomography (PET) is a nuclear medicine, functional imaging technique that produces a three-dimensional image of functional processes in the body. The system detects pairs of gamma rays emitted indirectly by a positron-emitting radionuclide (tracer), which is introduced into the body on a biologically active molecule.
- Positron emission tomography (PET) is being increasingly used for diagnosis, staging, and follow-up of various malignancies. It has been studied in the evaluation of various tumors including but not limited to solitary pulmonary nodules, nonsmall cell lung carcinoma, lymphoma, melanoma, breast cancer, and colorectal cancer.
- Computed tomography (CT) and magnetic resonance (MR) imaging rely on anatomic changes for diagnosis, staging, and follow-up of cancer. However, PET has the ability to demonstrate abnormal metabolic activity (at the molecular level) in organs that as yet do not show an abnormal appearance based on morphologic criteria. It aids in differentiation of malignant from benign lesions and in staging of malignancies.
- PET is also useful in the follow-up of patients following chemotherapy or surgical resection of tumor, most of whom have a complicating appearance at CT or MR imaging due to postoperative changes or scar tissue.

**Tumor physiology and radionuclide uptake**

- Most commonly used positron emitting radionuclide used in PET imaging is FDG [2-(fluorine-18) fluoro-2-deoxy-d-glucose], which is an analog of glucose.
- The hallmarks of malignant cells are rapid proliferation, increase in size, local invasion, and distant metastasis. Tumorigenesis is supported by numerous polypeptide growth factors [platelet derived growth factor (PDGF) and insulin-like growth factor] and factors promoting tumor angiogenesis [vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF)].

- Rapidly proliferating large tumors outgrow their vascular supply, resulting in ischemia and necrosis of the tumor. One to two mm is the limit of enlargement of tumor diameter beyond which vascularization is needed for further tumor growth, the 2-mm size limit representing maximal oxygen and nutrients diffusing distance capability from blood vessels.
- Malignant cells have increased glucose utilization due to upregulation of hexokinase activity. Glucose is taken up by tumor cells by facilitated transport [via glucose transporters (GLUT)] and then undergoes glycolysis with the formation of pyruvate under aerobic conditions. However, under hypoxic conditions (in a necrotic tumor), glucose is metabolized under anaerobic conditions with resultant increased tumor lactate levels.
- FDG is a radiopharmaceutical analog of glucose that is taken up by metabolically active tumor cells using facilitated transport similar to that used by glucose. The rate of uptake of FDG by the tumor cells is proportional to their metabolic activity.
- Like glucose, it undergoes phosphorylation to form FDG-6-phosphate; however, unlike glucose, it does not undergo further metabolism, thereby becoming trapped in metabolically active cells.

#### **Patient preparation and technique**

- Patients are required to fast for approximately 4–6 hours prior to PET to enhance FDG uptake by tumors as well as to minimize cardiac uptake.
- They are instructed to avoid caffeinated or alcoholic beverages but can have water during this period.
- Before injection of FDG, the blood glucose level is measured; a level of less than 150 mg/dL is desirable. Good control of blood glucose is essential because the uptake of FDG into cells is competitively inhibited by glucose, as they use a common transport mechanism [glucose transporters (GLUT)] for facilitated transport into both normal and tumor cells.
- There is lack of agreement as to administration of insulin in diabetic patients for glucose control. Insulin facilitates transfer of glucose into muscle, adipose, and several other tissues (except brain and liver tissue, which do not require insulin for efficient glucose uptake) via GLUT, and the administration of insulin for glucose control in diabetics may exaggerate physiologic uptake in muscles.
- Patients are also instructed to avoid any kind of strenuous activity prior to the examination and following injection of the radioisotope to avoid physiologic muscle uptake of FDG.
- Bowel cleansing, advocated by certain authors during PET, is not used prior to PET-CT at most centers.
- The typical dose of FDG is 10 mCi injected intravenously. Patient activity and speech are limited for 20 minutes immediately following injection of the radioisotope to minimize physiologic uptake by muscles. Imaging is initiated approximately 60 minutes following the injection of FDG.

#### **Interpretation of PET scan and SUV**

- There are different methods for assessment of radiotracer uptake by normal and pathologic tissues, such as visual inspection, the standardized uptake value (SUV), and the glucose metabolic rate.
- The most commonly used method is visual inspection in analysis of PET-CT results by comparing PET and CT data, as well as viewing fused PET-CT images.



- SUVs are used for semiquantification of FDG uptake.
- Another method of quantification of dynamic PET results is the more complex glucose metabolic rate calculation.
- The SUV is a semiquantitative assessment of the radiotracer uptake from a static (single point in time) PET image. The SUV of a given tissue is calculated with the following formula: **tracer activity in tissue/injected radiotracer dose/patient weight**, where tissue tracer activity is in microcuries per gram, injected radiotracer dose is in millicuries, and patient weight is in kilograms.

### Applications

- PET is both a medical and research tool. It is used heavily in clinical oncology (medical imaging of tumors and the search for metastases), and for clinical diagnosis of certain diffuse brain diseases such as those causing various types of dementias. PET is also an important research tool to map normal human brain and heart function, and support drug development.
- PET is also used in preclinical studies using animals, where it allows repeated investigations into the same subjects. This is particularly valuable in cancer research, as it results in an increase in the statistical quality of the data (subjects can act as their own control) and substantially reduces the numbers of animals required for a given study.

### Oncology

- PET scanning with the tracer fluorine-18 (F-18) fluorodeoxyglucose (FDG), called FDG-PET, is widely used in clinical oncology. Many other types of solid tumors will be found to be very highly labeled on a case-by-case basis—a fact that becomes especially useful in searching for tumor metastasis, or for recurrence after a known highly active primary tumor is removed.
- PET scan is used in oncology for detection of primary malignancies, multifocal disease, metastasis, differentiation of benign and malignant disease, detection of tumor recurrence during follow-up and differentiation of recurrence from radiation induced changes.

### Neuroimaging

- Neurology: PET neuroimaging is based on an assumption that areas of high radioactivity are associated with brain activity. What is actually measured indirectly is the flow of blood to different parts of the brain, which is, in general, believed to be correlated, and has been measured using the tracer oxygen-15.
- In practice, since the brain is normally a rapid user of glucose, and since brain pathologies such as Alzheimer's disease greatly decrease brain metabolism of both glucose and oxygen in tandem, standard FDG-PET of the brain, which measures regional glucose use, may also be successfully used to differentiate Alzheimer's disease from other dementing processes, and also to make early diagnosis of Alzheimer's disease. The advantage of FDG-PET for these uses is its much wider availability.
- PET imaging with FDG can also be used for localization of seizure focus: A seizure focus will appear as hypometabolic during an interictal scan.
- Several radiotracers (i.e. radioligands) have been developed for PET that is ligands for specific neuroreceptor subtypes. These agents permit the visualization of neuroreceptor pools in the context of a plurality of neuropsychiatric and neurologic illnesses.

- *Neuropsychology/cognitive neuroscience*: To examine links between specific psychological processes or disorders and brain activity.
- *Psychiatry*: Numerous compounds that bind selectively to neuroreceptors of interest in biological psychiatry have been radiolabeled with C-11 or F-18. Radioligands that bind to dopamine receptors (D1, D2, and reuptake transporter), serotonin receptors (5HT1A, 5HT2A, reuptake transporter) opioid receptors ( $\mu$ ) and other sites have been used successfully in studies with human subjects.

### Cardiology

- Cardiology, atherosclerosis and vascular disease study: In clinical cardiology, FDG-PET can identify so-called “hibernating myocardium”, but its cost-effectiveness in this role versus SPECT is unclear.
- FDG-PET imaging of atherosclerosis to detect patients at risk of stroke is also feasible and can help test the efficacy of novel anti-atherosclerosis therapies.

### Musculoskeletal imaging

- *Musculoskeletal imaging*: PET has been shown to be a feasible technique for studying skeletal muscles during exercises like walking. One of the main advantages of using PET is that it can also provide muscle activation data about deeper lying muscles such as the vastus intermedialis and the gluteus minimus, as compared to other muscle studying techniques like electromyography, which can be used only on superficial muscles (i.e. directly under the skin). A clear disadvantage, however, is that PET provides no timing information about muscle activation, because it has to be measured after the exercise is completed. This is due to the time it takes for FDG to accumulate in the activated muscles.

### Radiation exposure

PET scanning is noninvasive, but it does involve exposure to ionizing radiation.  $^{18}\text{F}$ -FDG, which is now the standard radiotracer used for PET neuroimaging and cancer patient management, has an effective radiation dose of 14 mSv.

## Q6. What is intravenous pyelography (IVP)? Describe its indications, contraindications and procedure.

### Discuss intravenous pyelography in brief.

**Ans.** IVP is a physiological study, which allows visualization of entire urinary tract. The study provides demonstration of renal parenchyma, pelvicalyceal system (PCS), ureters and urinary bladder, providing both anatomical and functional information.

### Indications

Many of the conditions evaluated by IVP in past are better evaluated by CT or MRI nowadays, hence there are limited indications of IVP in modern radiology.

- *Single shot IVP*: In case of blunt trauma abdomen with renal involvement, single shot IVP is undertaken to demonstrate the function of opposite kidney in case nephrectomy is considered. In single shot IVP, plain radiograph KUB region followed by 15 minute post-contrast film is obtained with sole purpose to look for contralateral kidney function.
- Early urinary tract tuberculosis.
- Early papillary necrosis.

- Early transitional cell carcinoma.
- Congenital anomalies of kidneys and ureters.
- To demonstrate involvement of ureters in advanced gynecological malignancies (carcinoma cervix and endometrium).
- Renal trauma (nowadays CECT abdomen is considered a modality of choice).
- As a work up of a live donor in renal transplant.
- After urological surgeries, e.g. ureteric surgeries.
- *Renal colic*: In case of renal stone, to look for exact position of calculus for ESWL, degree of hydronephrosis and residual renal function. DTPA scan is more sensitive and accurate for assessment of renal function with better quantification of parameters.
- To evaluate effect of surgery on urinary tract, e.g. pyeloplasty, ESWL, endourological therapy.

#### *Indications of pediatric IVP*

- An overview of urinary tract is needed, when other congenital anomalies are detected.
- A malformation of genitalia or other systemic malformation requires IVP as they are associated with genitourinary anomalies.
- Urinary tract obstruction: Pelviureteric junction obstruction (PUJO), congenital megaureter.
- Renal scarring is present or suspected because of recurrent UTI or VUR. A condition exists or is suspected that involves the upper and lower urinary tract such as Prune Belly syndrome or neurogenic bladder.

#### **Contraindications**

- History of previous serious reaction to injected iodinated contrast medium.
- Pregnancy and in women who indicates possibility of pregnancy.
- Impaired renal function (renal function should be checked by serum creatinine and urea level to avoid contrast-induced nephropathy).

#### **Procedure**

##### *Preparation of patient*

- Mild preparation of patient may make it easier to visualize small and faintly calcified renal stones and PCS.
- Low residue diet for 23 days prior to examination. An oral laxative, few hours before the examination reduces bowel gases and residual fecal matter in large bowel.
- Hydration should be maintained. Dehydration is unnecessary and overhydration should be avoided.
- Patient is asked to have empty stomach for at least 6–8 hours prior to procedure, to avoid chances of aspiration in case vomiting occurs as a reaction to injected contrast medium. Pediatric patients should take nothing by mouth for at least 3–4 hours.
- The patient should micturate prior to examination to avoid dilution of contrast in bladder.
- A quiet reassurance and informed consent is important.

##### *Contrast medium*

Contrast media are iodinated organic compounds used to opacify the renal parenchyma and urinary tract in IVP. They are injected intravenously, which get excreted in unchanged

manner through glomerular filtration and secretion into urinary tract leading to opacification of the same.

The quantity of contrast medium administered should be related to the weight of the patient (300 mg of iodine/kg of body weight). In adults, around 50 mL of contrast is injected. For infants and child, the dose is relatively higher because of poor concentrating ability of immature nephrons. Neonates and small children are given 600 mg of iodine/kg of body weight, maximum up to 20 mL of contrast medium.

It is important to look for several factors before injecting contrast medium:

- To check renal function (serum creatinine and blood urea). In case of compromised renal function, reduction in dose of contrast or use of less toxic (nonionic) contrast should be considered.
- Ask for the history of previous contrast medium injection (full dose should not be repeated within 72 hours) and any serious contrast reaction.
- Ask for the history of other allergic diseases (asthma, allergies to other drugs) which make patient prone for allergic reaction to contrast.
- Ask for history of several diseases like diabetes, sickle cell anemia, multiple myeloma, hypertension, and other renal diseases which make patient prone for contrast-induced nephropathy.
- Ask for history of other nephrotoxic drugs or drugs having exclusive renal excretion and serious adverse reaction (like metformin causing lactic acidosis).

Emergency tray fully equipped to manage anaphylactic reaction or minor contrast reaction should be ready within IVP room, prior to injection of contrast medium.

#### *Filming sequence*

- Noncontrast, plain KUB radiograph is obtained usually to look for bowel preparation, technical quality and exposure factor determination and to detect calcifications and calcific densities that can be calculi.
- Patient is made to lie in supine position on X-ray table, followed by intravenous injection of contrast medium. Abdominal binder is applied to compress the ureters and allow adequate distension of proximal ureter and PCS.
- First radiograph is obtained at 1 minute after contrast injection to look for nephrogram (renal parenchyma), however, in practice this film is usually omitted as renal outline is well visualized in 5 minute film.
- Five minute film to look for renal outline and PCS.
- 10 minute film immediately after release of compression, to look for entire length of ureters.
- 15 minute film in prone position, to demonstrate contrast in lower ureters and VUJ.
- Full bladder view when patient feels urge to micturate.

#### *Contraindications of abdominal compression*

- Abdominal aortic or iliac aneurysm
- Abdominal distension
- Acute abdominal pain
- Recent abdominal surgery
- Ureteral calculi

- Infants and young children
- Abdominal trauma, IVC filters
- In presence of urinary diversions and renal transplant.

At the end of procedure, the patient is kept under observation for at least 1 to 2 hours for possible contrast reactions.

### Complications of IVP

- *Adverse reaction to contrast:* Most reactions commence within 20 minutes of contrast injection.
- Spontaneous rupture of collecting system (forniceal rupture) in acute calculus disease.
- Contrast-induced nephropathy.

## Q7. Write short note on micturating cystourethrogram (MCU).

### Describe clinical indication, technique and contraindications of MCU.

**Ans.** Micturating cystourethrogram basically involves direct instillation of iodinated contrast into the urinary bladder and bladder is irradiated when filled with contrast and urethra is imaged when patient voids.

### Indications

#### Children

- In presence of urinary tract infections in infants and small children, MCU is performed to detect vesicoureteral reflux.
- *Suspected anatomic abnormalities of the bladder neck and urethra:* The examination may show posterior urethral valve as obstructing pathology in boys. Urethral strictures may be shown.
- Double urethra, urethral diverticulae.

#### Adults

- *Functional disorders of bladder and urethra:* A complete urodynamic assessment is indicated. This involves pressure flow studies together with cystography.
- Suspected vasicovaginal and vesicocolic fistula.
- *Suspected bladder trauma:* Cystography can confirm bladder rupture. It may also be useful postoperatively in evaluating a suspected bladder leak.

### Contraindications

- *Urinary tract infection:* Appropriate antibiotics are prescribed to clear any infection before cystography is performed.
- *Hypersensitivity to contrast media:* A relative contraindication as there is some systemic absorption of the contrast medium from the bladder.

### Technique

- The procedure is explained to patient or parents and informed consent is obtained.
- The examination is most commonly performed via perurethral Foley's catheter or small bore feeding tube (6 or 8F).
- The catheter specimen of urine is collected and sent for culture and microscopy. The catheter is tapped to thigh and patient is then brought to examination room. Residual urine is drained from the bladder.

- Any excess tape should be removed prior to the introduction of contrast so that the catheter may be removed quickly once voiding has begun.
- Adequately diluted, water soluble contrast medium is injected through catheter into the urinary bladder under fluoroscopic guidance. Intermittent fluoroscopy is performed to look for the bladder filling and vesicoureteral reflux if any.
- A spot radiograph is obtained during early filling phase of urinary bladder as this is the optimal time to demonstrate the ureterocolic. Spot radiographs are taken if any reflux is detected.
- Urinary bladder is filled till patient complains of feeling of fullness or the patient is seen to start voiding in case of pediatric patient.
- The catheter is then removed and spot radiographs are obtained when patient micturates on absorption paper on table. Images should include oblique views of bladder, ureter and urethra during voiding together with a post void view to include kidneys. Lateral views are obtained to demonstrate vascovaginal or vesicocolic fistulas.
- The timing of catheter withdrawal is important to satisfactory imaging of the voiding phase.
- If child does not micturate immediately, a long wait may follow. Bladder emptying may be incomplete due to the effects of sedation, because of dysuria following catheterization, in case of neurogenic bladder, or because of refilling from above where there is significant reflux. Occasionally, it is necessary to manually compress the suprapubic region for emptying of bladder.

#### **Alternative technique**

- *Suprapubic bladder puncture:* In infants, this approach may be used sometimes in case of lower urinary tract obstruction. In adults, pelvic trauma is the most common indication for a suprapubic approach. In many of these cases, it has been impossible to pass a catheter per urethrum or an elective decision has been made to insert a suprapubic catheter because injury to bladder or urethra is suspected.
- *Urethrocystography:* Contrast medium may be instilled into the urinary bladder during retrograde urethrography. Indeed, this may be necessary to adequately visualize posterior urethra.
- *Following IVU:* This technique is no longer widely employed but imaging of micturition after bladder has become uncomfortably full at the end of an IVP allows some basic questions to be answered.

#### **Complications**

- *Urinary tract infection:* The incidence of UTI is approximately 10 to 30%. Children are routinely given antibiotic cover for that.
- Complications of catheterization include urethral trauma, bladder trauma, and insertion of a suprapubic catheter into the peritoneal cavity or extravasical space. Trauma can cause transient hematuria, dysuria, urinary frequency, urinary retention.
- Complications of contrast media include adverse reaction due to absorbed contrast, contrast induced cystitis.

**Q8. What is ERCP? Enumerate the indications and complications with brief note of procedure of ERCP.**

**Ans.** Endoscopic retrograde cholangiopancreatography (ERCP) is a technique that combines the use of endoscopy and fluoroscopy to diagnose and treat certain problems of the biliary or pancreatic ductal systems. Through the endoscope, the physician can see the inside of the stomach and duodenum, and inject radiographic contrast into the ducts in the biliary tree and pancreas so they can be seen on X-rays.

ERCP is used primarily to diagnose and treat conditions of the bile ducts and main pancreatic duct, including gallstones, inflammatory strictures (scars), leaks (from trauma and surgery), and cancer. ERCP can be performed for diagnostic and therapeutic reasons, although the development of safer and relatively noninvasive investigations such as magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasound has meant that ERCP is now rarely performed without therapeutic intent.

### Indications

#### *Diagnostic*

- *Obstructive jaundice:* This may be due to several causes.
- *Chronic pancreatitis:* A now controversial indication due to widespread availability of safer diagnostic modalities including endoscopic ultrasound, high-resolution CT, and MRI/MRCP.
- Gallstones with dilated bile ducts on ultrasonography.
- Bile duct tumors.
- Suspected injury to bile ducts either as a result of trauma or iatrogenic.
- Sphincter of Oddi dysfunction.
- Pancreatic tumors no longer represent a valid diagnostic indication for ERCP unless they cause bile duct obstruction and jaundice. Endoscopic ultrasound represents a safer and more accurate diagnostic alternative.

#### *Therapeutic*

Any of the above when the following may become necessary:

- Endoscopic sphincterotomy (both of the biliary and the pancreatic sphincters)
- Removal of stones
- Insertion of stent(s)
- Dilation of strictures (e.g. primary sclerosing cholangitis, anastomotic strictures after liver transplantation).

### Contraindications

- Acute pancreatitis (unless persistently raised or worsening bilirubin suggests ongoing obstruction)
- Previous pancreatoduodenectomy
- Coagulation disorder if sphincterotomy planned
- Recent myocardial infarction
- Inadequate surgical back-up
- History of contrast dye anaphylaxis
- Poor health condition for surgery
- Severe cardiopulmonary disease.

**Procedure**

- The patient is sedated or anesthetized. Then a flexible camera (endoscope) is inserted through the mouth, down the esophagus, into the stomach, through the pylorus into the duodenum where the ampulla of Vater (the opening of the common bile duct and pancreatic duct) exists.
- The sphincter of Oddi is a muscular valve that controls the opening of the ampulla. The region can be directly visualized with the endoscopic camera while various procedures are performed.
- A plastic catheter or cannula is inserted through the ampulla, and radiocontrast is injected into the bile ducts and/or pancreatic duct. Fluoroscopy is used to look for blockages, or other lesions such as stones.
- When needed, the opening of the ampulla can be enlarged (sphincterotomy) with an electrified wire (sphincterotome) and access into the bile duct obtained so that gallstones may be removed or other therapy performed.
- Other procedures associated with ERCP include the trawling of the common bile duct with a basket or balloon to remove gallstones and the insertion of a plastic stent to assist the drainage of bile. Also, the pancreatic duct can be cannulated and stents be inserted. The pancreatic duct requires visualization in cases of pancreatitis.
- In specific cases, a second camera can be inserted through the channel of the first endoscope. This is termed duodenoscope-assisted cholangiopancreatography (DACP) or mother-daughter ERCP. The daughter scope can be used to administer direct electrohydraulic lithotripsy to break up stones, or to help in diagnosis by directly visualizing the duct (as opposed to obtaining X-ray images).
- The gallbladder should be surgically removed (cholecystectomy) following successful removal of gallstones from the bile ducts.

**Risks**

- The major risk of an ERCP is the development of pancreatitis, which can occur in up to 5% of all procedures. This may be self-limited and mild, but may require hospitalization, and rarely, may be life-threatening. Patients at additional risk for pancreatitis are younger patients, patients with previous post-ERCP pancreatitis, females, procedures that involve cannulation or injection of the pancreatic duct, and patients with sphincter of Oddi dysfunction.
- Gut perforation is a risk of any endoscopic procedure, and is an additional risk if a sphincterotomy is performed. As the second part of the duodenum is anatomically in a retroperitoneal location (that is, behind the peritoneal structures of the abdomen), perforations due to sphincterotomies are also retroperitoneal. Sphincterotomy is also associated with a risk of bleeding.
- Oversedation can result in dangerously low blood pressure, respiratory depression, nausea, and vomiting.
- There is also a risk associated with the contrast dye in patients who are allergic to compounds containing iodine.
- Other complications (less than 1 per 100) may include; heart and lung problems, bleeding after sphincterotomy, infection in the bile duct (cholangitis) and perforation (a tear in the intestine). In some rare cases, ERCP can even cause fatal complications.



**Q9. What is MRCP? Discuss in brief and compare with ERCP.****What is ERCP and MRCP? Compare ERCP and MRCP with advantages and disadvantages of both.**

**Ans.** Magnetic resonance cholangiopancreatography has got a widespread clinical acceptance and has almost replaced diagnostic ERCP. MRCP visualizes biliary and pancreatic tree non-invasively without any contrast injection or radiation.

**Technique**

- *Preparation:* Patient should be empty stomach for at least 6–8 hours to avoid any fluid in GI tract especially in stomach. If fluid is present in stomach, it can be suppressed by barium or blue berry juice.
- MRCP includes heavily T2-weighted sequences that show stationary or slow moving fluid such as bile as high signal intensity. A number of techniques have been employed to achieve heavily T2 weighting, however, two techniques commonly used include RARE and HASTE.
  - RARE (Rapid acquisition relaxation enhancement)
  - HASTE (Half-Fourier acquisition single-shot turbo spin-echo)
- MRCP should also include depending on the case, other MR sequences to evaluate extraductal structures and pathologies.
- MRCP can also be done after intravenous injection of hepatobiliary specific gadolinium contrast agents (Gd BOPTA, Gd EOB DTPA, Mn DPDP), followed by delayed T1W sequence depending on the type of the agent injected.

**Applications of MRCP***Cystic diseases in the biliary tract*

- MRCP is as effective as ERCP in evaluating choledochal cyst, choledochocoele and Caroli's disease.

*Congenital anomalies of pancreas and biliary tree*

- *Pancreatic divisum:* MRCP is superior to ERCP in detecting pancreatic divisum. Congenital variations like low insertion of cystic duct, parallel course of cystic duct, medial cystic duct insertion and aberrant right hepatic duct are visualized on MRCP. Detection of these variations is important to aid complications during cholecystectomy especially laparoscopic.
- *Biliary atresia:* It can noninvasively establish the diagnosis of biliary atresia.

**Choledocholithiasis**

Accurate diagnosis of stone in CBD is very important before cholecystectomy; MRCP is an excellent method to detect those stones, comparable to ERCP and superior to other modalities like USG and CT.

**Primary sclerosing cholangitis**

- It is characterized by multiple irregular strictures and saccular dilatations of the intrahepatic and extrahepatic bile ducts producing beaded appearance.
- MRCP is useful in the diagnosis and follow-up of primary sclerosing cholangitis. ERCP may result in progression of cholestasis and may not show duct proximal to severe stenosis.

**Postsurgical complications**

- Postsurgical complications like benign strictures, retained stones, biliary leak and biliary fistula are effectively evaluated with MRCP.

**Chronic pancreatitis**

- Chronic pancreatitis is characterized by pancreatic duct dilatation, narrowing or stricture and irregularity.
- Alcoholic chronic pancreatitis is usually heterogeneous and characterized by side branch dilatation and ductal calcifications whereas obstructive pancreatitis is more homogenous, lack calcification and are associated more often with main duct dilatation.
- MRCP is useful for detecting chronic pancreatitis and for identification of surgically or endoscopically correctable lesions.

**Neoplastic lesions**

- MRCP can show duct proximal to obstruction caused by neoplasms like cholangiocarcinoma or pancreatic head carcinoma.
- Conventional MR imaging like fat saturated post-contrast T1 weighted images should be combined with MRCP for the evaluation of extent and spread of the lesion.

**Secretin MRCP (s-MRCP)**

- Secretin stimulates exocrine pancreatic secretions and distends pancreatic duct.
- Secretin MRCP performed after administration of secretin is useful for functional imaging and improved anatomic depiction of pancreatic duct system. It reduces the false positive depiction of strictures.
- The main limitation of s-MRCP is the high cost of secretin.

**Comparison of MRCP and ERCP**

MRCP	ERCP
• Noninvasive, radiation free	• Involves contrast injection and radiation
• Produces images of the duct in natural physiological state	• Ducts are distended with contrast
• Can be combined with conventional MR sequences to evaluate extraductal structures	• Extraductal pathologies and structures cannot be assessed
• Ducts beyond obstruction can also be assessed	• Ducts proximal to the obstruction may not be seen if obstruction is complete
• Nonoperator dependent	• Operator dependent
• Safe	• Involves morbidity and mortality. Complications include pancreatitis, sepsis, perforation and hemorrhage
• It can be useful after incomplete or unsuccessful ERCP	• Up to 10% technical failure are reported in ERCP
• It can be performed in postsurgical patients in which biliary enteric anastomosis is performed	• It is not possible clinically to perform ERCP in postoperative patients

Contd...

Contd...

MRCP	ERCP
<ul style="list-style-type: none"> <li>Major limitation is lack of therapeutic capability</li> </ul>	<ul style="list-style-type: none"> <li>Therapeutic options like endoscopic lithotomy, brush cytology, sphincterotomy, collection of pancreatic juice, stricture dilatation, stent placement and biopsy are possible with ERCP</li> </ul>
<ul style="list-style-type: none"> <li>Second limitation is its limited spatial resolution</li> </ul>	<ul style="list-style-type: none"> <li>Higher spatial resolution is achievable with ERCP may be important in presence of pancreatic ductal dilatation</li> </ul>

**Q10. Discuss in brief the role of radionuclide imaging in various musculoskeletal disorders.**

**Write a short note on radioisotope bone scan.**

**Role of radionuclide imaging in detection of skeletal metastases.**

**Ans.** Musculoskeletal system is a dynamic organ system which responds to systemic as well as localized stress and hence apart from anatomical visualization, it needs physiological evaluation as well. The major advantage of the nuclear medicine imaging comes from the fact that functional changes appear much more before than anatomical changes. Hence, nuclear imaging allows early detection of diseases affecting bones.

**Bone scintigraphy**

- In spite of newer development in field of nuclear medicine, bone scintigraphy remains the most commonly employed method for musculoskeletal imaging.
- It is done with the intravenous injection of bone seeking radiopharmaceuticals. These compounds are usually phosphates labeled with  $^{99m}\text{Tc}$ . The most widely used is  $^{99m}\text{Tc}$  – methylene diphosphonate (MDP). Others are  $^{99m}\text{Tc}$  – ethylene hydroxy diphosphonate (EHDP) and  $^{99m}\text{Tc}$  – hydroxy methylene diphosphonate (HDP). The presence of p-c-p bond makes these compounds resistant to hydrolysis by bone phosphatases, thereby increasing the biological half-life.
- Three to four hours after the intravenous injection, 25 to 35 percent of the radiotracer is retained in the normal adult skeleton; the rest is excreted in the urine via kidney.
- These phosphorus-containing compounds attach themselves to hydroxyapatite crystal surface by the process of chemisorptions. Thus, they are localized at the site of direct and indirect osteoblastic activation. This osteoblastic activation can be due to wide range of pathologies, therefore, reducing the specificity.

*Protocol*

- Although protocols vary among institutions, imaging is typically performed 2–6 hours after intravenous administration of 740–925 MBq (20–25 mCi) of  $\text{Tc}^{99m}$ -labeled diphosphonates. The delay between injection and imaging allows clearance of the radiotracer from the soft tissues, resulting in a higher target-to-background ratio and improved visualization of bone.
- Skeletal detail can be further enhanced by encouraging patients to drink copious amounts of fluid after radiotracer injection.

- A gamma camera equipped with a low-energy, high-resolution collimator will yield the highest resolution images. Additional anterior and posterior whole-body images are often obtained as needed. In addition dynamic flow and pool images are obtained in selected cases.

### **SPECT and SPECT CT**

- Given the lack of anatomical information provided by bone scan especially in regions of complex anatomy, additional method re-employed.
- SPECT provides three-dimensional pictures and is beneficial in patients with normal planar images despite symptoms and in those with equivocal findings.
- It is most useful for evaluation of thoracolumbar spine, pelvis and skull. These areas have extensive surrounding soft tissue and complicated body contours, and thus superior image contrast provided by SPECT improves lesion detection.
- However, even with SPECT correlation with anatomic imaging is needed in many cases. SPECT CT imaging with hybrid cameras has been introduced to overcome this shortcoming and to provide both anatomic and functional imaging.

### **PET and PET CT**

- Nowadays PET CT is being increasingly used for the imaging of primary and secondary tumors of bones and infections.
- In addition to  $^{18}\text{F}$ -FDG, other tracer for bone PET is  $^{18}\text{F}$ -Fluoride. It can also be used for quantitative studies of skeletal disorder. The mechanism of uptake in bone is quite similar to MDP that is absorption onto the bone surface with the predilection for the sites of the bone formation. The uptake of  $^{18}\text{F}$ -Fluoride is two-fold higher and its blood clearance is significantly faster compared with the  $^{99\text{m}}\text{Tc}$ -MDP, resulting in an increased bone to background ratio.
- In addition, PET offers high sensitivity and high resolution, and therefore enables to perform highly accurate whole body screening for metastasis.

### **Normal scintigraphic findings**

- There is symmetric distribution of activity throughout the skeletal system in healthy adults. Urinary bladder activity, faint renal activity, and minimal soft-tissue activity are also normally present.
- In children, intense symmetric uptake in the physes of the long bones, which represent centers of normal growth and hematopoietic production, is typically present. The marrow-containing flat facial bones also demonstrate accumulation of radiotracer in children.
- The accumulation of radiotracer in bone generally decreases with age. However, there are sites of persistently increased symmetric uptake, such as the acromial and coracoid processes of the scapulae, the medial ends of the clavicles, the junction of the body and manubrium of the sternum (angle of Louis), and the sacral alae.
- Increased radiotracer accumulation in the jaw may be due to dental disease or to malocclusion of dentures.
- Symmetric areas of increased calvarial activity occur in hyperostosis frontalis.
- In the neck, activity in calcified thyroid cartilage and in the apophyseal joints of the cervical vertebrae in patients with asymptomatic degenerative changes can also be seen.

### Applications of radionuclide bone imaging

#### *Metastasis*

- Many if not most bone scans are performed in patients with a diagnosis of malignancy, especially carcinoma of the breast, prostate gland, and lung. About 75% of patients with malignancy and pain have abnormal bone scintigraphic findings. Perhaps even more importantly, 25–45% of asymptomatic patients with malignancy have scintigraphic evidence of bone metastases.
- The usual pattern consists of increased radiotracer deposition in areas of osteoblastic reparative activity in response to tumor osteolysis. The presence of multiple, randomly distributed areas of increased uptake of varying size, shape, and intensity are highly suggestive of bone metastases.
- Although multiple foci of increased activity may be encountered in other pathologic conditions, it is often possible to distinguish metastatic disease from other entities by analyzing the pattern of distribution of the abnormalities.
- When the metastatic process is diffuse, virtually all of the radiotracer is concentrated in the skeleton, with little or no activity in the soft tissues or urinary tract. The resulting pattern, which is characterized by excellent bone detail, is frequently referred to as a **superscan**. A superscan may also be associated with metabolic bone disease. Unlike in metastatic disease, however, the uptake in metabolic bone disease is more uniform in appearance and extends into the distal appendicular skeleton. Intense calvarial uptake that is disproportionate to that in the remainder of the skeleton is another feature of a metabolic superscan.
- Metastatic disease occasionally manifests as a solitary abnormality, usually in the spine. Degenerative changes may also manifest as an isolated abnormality. Single photon emission computed tomography (SPECT) is useful for differentiating between these two pathologic conditions.
- To accurately interpret radionuclide bone images obtained in patients with tumor, one must be cognizant of the effects that treatment can have on the study.

#### *Trauma*

- Although most fractures are detected radiographically, bone scintigraphy is useful for detecting fractures in patients with a history of trauma and equivocal or frankly negative radiographs. Most fractures are scintigraphically detectable within 24 hours of their occurrence; however, in elderly patients with osteopenia and unremarkable findings at initial bone scintigraphy performed within 24 hours of injury, repeat scintigraphy may be performed at 72 hours to maximize sensitivity.
- The minimum time for normalization of activity is 6 months after the fracture, and nearly all fractures will show normalization of activity by 2 years. Because remodeling is an ongoing process in nonaligned fractures, these fractures may never attain a normal scintigraphic appearance.
- In athletic individuals, the lower extremity is often the site of musculoskeletal trauma. Commonly seen conditions include enthesopathies, stress fractures, and “shin splints.”
- Plantar fasciitis, a form of localized reactive periostitis, develops in individuals engaged in activities that involve extensive foot dorsiflexion, such as running and aerobics. Pain and tenderness are concentrated at the site of the insertion of the long plantar tendon into

the inferior aspect of the calcaneus. The typical appearance of this entity on radionuclide bone images is that of focally increased activity, which may be intense, at the site of the tendon insertion.

- Radionuclide bone imaging is often used to differentiate tibial stress fractures from shin splints. Hyperperfusion and hyperemia are typically present in acute stress fracture. Delayed images demonstrate focal fusiform uptake in the lesion; this uptake often occurs at the junction of the middle and distal thirds of the tibia. Shin splints are due to excessive exertion of the tibialis and soleus muscles of the legs, which gives rise to periostitis at the tibial insertions of these muscles. Unlike in stress fractures, angiograms and blood pool images are usually normal in shin splints. Delayed bone images reveal longitudinally oriented linear areas of increased uptake of varying intensity that involve one-third or more of the posterior tibial cortex. The differentiation of stress fracture from shin splints is important because their treatments are very different.

### **Infection**

- Three-phase bone scanning has an accuracy of over 90% and is the radionuclide procedure of choice for diagnosing osteomyelitis in bones not affected by underlying conditions.
- The first (dynamic) phase reflects the relative amount of blood flow to the area of interest, whereas the second (blood pool) phase reflects the amount of activity that has extravasated into the tissues around the area of interest. The third (delayed) phase reflects the rate of bone turnover.
- The classic appearance of osteomyelitis on three-phase bone scan consists of focal hyper-perfusion, focal hyperemia, and focally increased bone uptake.
- Abnormalities at radionuclide bone imaging reflect increased bone mineral turnover in general, not infection specifically. Therefore, conditions associated with increased bone mineral turnover (e.g. tumors, fractures, joint neuropathy) may mimic osteomyelitis at three-phase bone scintigraphy.
- Under these circumstances, three-phase bone imaging is less useful, primarily because of diminished specificity. To improve specificity, complementary imaging with gallium-67 citrate (for spinal infection) or indium-111-labeled autologous leukocytes (for the appendicular skeleton) is often performed.

Other applications of bone scan in rare disorders include Paget disease, reflex sympathetic osteodystrophy, hypertrophic osteoarthropathy, avascular necrosis, spondylolysis, etc.

### **Q11. Enumerate the radionuclide isotopes for thyroid imaging. Describe the role of radionuclide imaging in various thyroid disorders.**

#### **Discuss in brief the technique and isotopes of thyroid scan.**

- Ans.** Nuclear medicine plays an important role in imaging of thyroid gland, providing functional information that complements anatomical information obtained from other radiological investigation.

### **Indications**

#### *Diagnostic*

- In thyrotoxicosis, to distinguish between diffuse toxic goiter and autonomous toxic nodule.
- Evaluation of dominant solitary thyroid nodule.

- To find ectopic, lingual or mediastinal thyroid.
- Detection and staging of post-operative thyroid cancer.
- Evaluation of neonatal hypothyroidism.
- To monitor the progress of thyroiditis.

#### *Therapeutic*

- Treatment of hyperthyroidism.
- Treatment of differentiated thyroid cancer.

#### **Radionuclide used in thyroid imaging**

*<sup>99m</sup>Tc-Pertechnetate: most widely used agent for thyroid imaging*

- It is taken up by the thyroid gland in the same manner as iodine (active transport). After trapping pertechnetate slowly “washes off” from the gland, it does not undergo organification. It is excreted unchanged by kidneys, salivary glands and GIT.
- *Dose:* 3–10 mCi.
- Imaging is performed after 15–20 minutes after intravenous injection as peak thyroid activity occurs at this time.

#### **Advantages**

- Low cost, reduced radiation exposure and scans can be completed in a much shorter time.
- Greater photon flux than iodine so that detectability of small lesions is improved.
- Can be used even when the patient is taking thyroid blocking drugs.
- Excellent physical characteristics.

#### **Disadvantages**

- Target to background ratio is less favorable.
- Some lesions can show iodine pertechnetate discordance (hot on pertechnetate scans and cold on iodine scan)—need further imaging.

#### *Iodine 123 (<sup>123</sup>I)*

- Its half-life is 13.6 hours and it decays by electron capture.
- Absolutely contraindicated in pregnancy. Breastfeeding is relative contraindication to thyroid scanning as 20% of an administered dose is excreted in breastmilk.
- 200–400 mCi of the agent is given orally 24 hours prior to imaging. Images are typically acquired 4 hours following administration of the tracer and uptake values are determined at 4 and 24 hours.
- Its advantage is having low radiation dose and short half-life.
- Its disadvantages include high cost, time consuming and limited availability. It cannot be used in patients taking antithyroid drugs.

#### *Iodine 131 (<sup>131</sup>I)*

- It has half-life of 8 days and emits a high energy gamma and a wide spectrum of beta particles.
- Indications
- The long half-life, high energy gamma and beta emission limit the usefulness of <sup>131</sup>I for imaging purposes. Its administration results in very high radiation dose to the thyroid, 90% of which result from beta decays.

- It is the tracer of choice for whole body scintigraphy for metastasis from thyroid carcinoma.
- Because of the beta emission, it has high therapeutic uses.

### Others agents for thyroid imaging

#### *Thallium 201 ( $^{201}\text{Tl}$ )*

- The main clinical application is in patient with residual or recurrent thyroid cancer with raised thyroglobulin and a negative  $\text{I}^{131}$  body surveys. In 29% of such patients, thallium scan can detect metastasis.

#### *$^{99\text{m}}\text{Tc}$ -MIBI and $^{99\text{m}}\text{Tc}$ -tetrofosmin*

- Their role is similar to thallium in thyroid imaging. MIBI images have better resolution and are easy to interpret than thallium scan.
- Thallium, MIBI and tetrofosmin do not require discontinuation of antithyroid drugs.

**Medullary carcinoma thyroid:**  $^{123}\text{I}$ -MIBG, pentavalent  $^{99\text{m}}\text{Tc}$ -DMSA,  $^{111}\text{In}$ -Pentetreotide

### Imaging patterns in various thyroid disorders

#### *Reduced or no uptake of radiotracer*

- Blocked trapping function: Iodine load from administration of iodine containing drugs or contrast media.
- Administration of exogenous thyroid hormone.
- Blocked organification: Due to administration of propylthiouracil.
- Diffuse parenchymal destruction: In subacute or acute thyroiditis.
- Hypothyroidism: Congenital or post-surgical.

#### *Increased uptake of radiotracer*

- Thyroiditis: Early phase of Hashimoto's thyroiditis.
- Hyperthyroidism: Diffuse or nodular.
- Rebound after withdrawal of antithyroid medication.
- Iodine starvation.
- Low serum albumin.
- Lithium therapy.

### Thyroid nodules

- The nodule may be cold or hot depending on uptake of tracer in nodule in comparison to rest of the thyroid gland.
- Most of the hot nodules are TSH independent autonomous toxic nodules. Cold nodules can be malignant or benign, which require further investigation and FNAC for characterization.

### Specific conditions

- *Graves' disease:* Scintigraphy demonstrates diffusely enlarged thyroid gland showing increased activity throughout the gland. Pyramidal lobe may also be seen enlarged.
- Toxic autonomous nodule (Plummer disease): Scan can demonstrate hyperfunctioning nodule, since it concentrates the radiopharmaceutical to a greater degree than the surrounding extranodular thyroid tissue which is suppressed.



- *Toxic multinodular goiter*: Multiple nodular areas of increased uptake are seen giving heterogeneous appearance.
- *Hypothyroid states*: In neonates, with suspected primary congenital hypothyroidism, thyroid scan helps to confirm presence of a normal gland in patients and helps to differentiate three subgroups of primary congenital hypothyroidism: 1) non-visualization of normal functional thyroid tissue, 2) hypoplastic or ectopic glands, 3) dyshormonogenesis.
- *Hashimoto's thyroiditis*: low uptake with poor visualization of the gland is common. Patchy tracer distribution may be seen. Pyramidal lobe enlargement can also be observed.
- *DeQuervain's thyroiditis*: Patchy tracer uptake is noted in enlarged and tender thyroid gland.
- *Thyroid carcinoma*: The incidence of differentiated thyroid cancer in hypofunctioning nodule is directly proportional to the iodine supply in a given population. The better the iodine supply, higher the probability of a hypofunctioning nodule being malignant. Papillary carcinoma usually concentrates radioactive iodine. Follicular carcinoma usually accumulates pertechnetate but fails to accumulate iodine, producing discordance. Anaplastic carcinoma may not show any activity. Medullary carcinoma does not take up iodine or pertechnetate. Other agents mentioned above may get accumulated in medullary carcinoma thyroid.
- The other major indication of thyroid scanning in patient with thyroid malignancy is to detect and localize metastasis with whole body studies. Well-differentiated recurrences and metastasis will have ability to take up iodine and pertechnetate.  $I^{131}$  whole body scan can identify most functioning metastasis, which is usually seen in lungs, neck and bones. However, less than third of metastasis is less well-differentiated and show uptake of thallium, MIBI or tetrofosmin. PET with  $^{18}F$  FDG may also have role in follow-up of patients with thyroid cancers.

**Q12. What are the radionuclide isotopes used for renal imaging? Describe the role of radionuclide imaging in various renal disorders.**

**Discuss the role of renal scan for various renal diseases.**

**DMSA and DTPA scan.**

**Ans.** Renal structure can be investigated in great detail using X-ray methods, ultrasound and MRI, but functional evaluation of renal and urinary tract requires additional need of radionuclide imaging. Besides being highly sensitive, these techniques are nontoxic, simple, rapid and noninvasive and allow acquisition of information often unavailable from other sources.

Using these techniques, it has been now possible to calculate the split renal function in various renal disorders, evaluate kidney size in advanced renal azotemia, detect presence of kidneys which are poorly functioning, evaluate renal perfusion, obtain a differential diagnosis of the upper urinary tract dilatation, diagnose urinary tract obstruction, detect intrarenal space occupying lesions and monitor the renal function in renal failure.

**Radionuclide agents**

The various radiopharmaceuticals used for the assessment of kidney are broadly divided into two groups:

- The first group includes those that are rapidly excreted by kidneys and thus enable evaluation of renal function and urinary drainage.  $^{99m}Tc$ -LLEC,  $^{99m}Tc$ -MAG3,  $^{99m}Tc$ -DTPA,

$^{99m}\text{Tc}$ -GHA and  $^{123}\text{I}$ -OIH are included in this group, with the exception of GHA; these agents are not used for static cortical scintigraphy.

- The second group includes agents, which are concentrated in the renal parenchyma for a sufficiently long time, thus enabling detailed mapping of renal parenchyma. Included in this group are  $^{99m}\text{Tc}$ -DMSA and  $^{99m}\text{Tc}$ -GHA.
- Note that GHA is included in both of these groups of agents because approximately 10–15% of the injected dose is retained in the renal parenchyma while 65% of the injected dose is eliminated in the urine within 6 hours after injection.

Various radionuclide imaging methods used for assessment of urinary tract are broadly divided into four categories:

### **Dynamic renography**

This refers to serial continuous imaging following intravenous injection of radio-pharmaceutical. It encompasses three principle techniques: Dynamic renography, diuretic renography and captopril renography.

#### *1. Dynamic renography provides the following information:*

- Relative and absolute size of functional renal units.
- Total renal function (kidney/background ratio).
- Renal or split renal function.
- Overall renal morphology and redistribution of renal parenchyma.
- Position of renal units.

#### *2. Diuretic renography*

- Furosemide causes rapid diuretic response that drains out the tracer from dilated nonobstructed system. In significant outflow obstruction, tracer in the real area may decrease slowly or fail to decrease or even increase in response to a diuretic challenge.

#### *3. Captopril renography*

- Captopril renography is used for assessment of renal artery stenosis patients.
- Captopril blocks the formation of angiotensin II, producing dilatation of efferent arterioles and a fall in transcapillary pressure gradient. This causes a significant fall in GFR in patient with renal artery stenosis that can be easily diagnosed by renography.

### **Renal cortical scintigraphy**

- It includes scanning by DMSA, which is used mainly for assessment of cortical scarring in pyelonephritis and vesicoureteral reflex (VUR). Various congenital anomalies can also be evaluated by this method.

### **Radionuclide cystography and voiding cystography**

- This has been accepted as technique of choice for the follow-up of male child with urinary tract obstruction and VUR and as a primary diagnostic modality for female child and sibling screening of a male child.
- The estimated gonadal radiation dose is one-hundredth of MCU.
- It is more sensitive than MCU in detecting VUR.
- Other parameters like residual urine volume, bladder volume at the time of reflux and the rate of clearance of refluxed urine can also be calculated.

- The only disadvantage of radionuclide cystography is poor anatomical resolution of bladder and urethra.

### **Clearance studies for the assessment of renal function: GFR estimation, EPRF estimation**

- Many radiopharmaceuticals are used to calculate GFR, these include  $^{99m}\text{Tc}$ -DTPA and  $^{131}\text{I}$ -iothalamate. In India,  $^{99m}\text{Tc}$ -DTPA is mainly used for GFR estimation.
- Effective plasma renal flow (EPRF) can also be used by use of  $^{131}\text{I}$ -OIH and  $^{99m}\text{Tc}$ -MAG.

### **Applications**

#### *Urinary tract obstruction*

- Radionuclide studies may be necessary to diagnose obstruction, to ascertain its site or level, to determine the extent and severity or to measure how well the obstructed kidney is functioning.
- In the differential diagnosis of obstruction, diuretic renography is of immense help especially in those patients with a nonobstructed dilated collecting system associated with indeterminate flow curves.

#### *Urinary tract infection*

- $^{99m}\text{Tc}$ -DMSA is very useful in identification of renal damage due to early pyelonephritis. Even in the absence of any structural abnormality on ultrasound and CT, DMSA scan can show focal abnormality in uptake of the tracer.
- DMSA scan can also be used to quantify the degree of renal damage, to follow the progress of antibiotic treatment and to assess recovery or residual damage.
- Imaging with DMSA can help detect cortical scarring of pyelonephritis. In the detection of cortical scarring, DMSA scan is more sensitive and specific than intravenous urogram (IVU).
- Besides, in patients with chronic pyelonephritis with uncontrolled hypertension, demonstration of a severe segmental scar on a DMSA scan may guide the surgeon in undertaking partial nephrectomy.

#### *Congenital anomalies*

- Renal radionuclide scanning is helpful in functional and structural evaluation of various congenital anomalies of kidneys.
- It helps in management and accurate assessment of anomalies in case of polycystic kidneys, medullary sponge kidney, horseshoe kidney, crossed ectopias, malrotated kidneys, etc.
- Preoperative and serial postoperative morphological and functional evaluation of kidneys, upper urinary tract and urinary bladder can be effectively done in patients with posterior urethral valve.

#### *Renovascular hypertension*

- A major advance in the diagnosis and management of RVH is the advent of captopril renal scintigraphy.
- The concept of using captopril in diagnosis of RAS arises from the observation that patients with bilateral renal artery stenosis or patient with solitary kidney having renal artery stenosis had a tendency to develop acute renal failure when treated with these drugs.

- Captopril acts to block the step where angiotensin I is converted to angiotensin II, and as a result prevent subsequent increase in the efferent arteriolar tone. In the absence of this compensatory mechanism, GFR drops significantly in affected kidney.
- A normal baseline study followed by unilateral decreased in split renal function are the hallmark of unilateral renal artery stenosis.

#### *Renal failure*

- Radionuclide clearance studies provide valuable information and help in monitoring glomerular and tubular function in nephropathies of various origins, the most significant being the diabetic nephropathy.
- The prospective monitoring of the diminished renal function in diabetic patients should include accurate estimation of GFR by various radionuclide imaging combined with fractional clearance of high molecular weight proteins. This is of particular importance when treatment monitoring in early proteinuric phase of diabetic nephropathy is done.
- Renal transplant evaluation.
- Radionuclide imaging provides rapid, effective and noninvasive method of detecting many of the ischemic, immunological and mechanical complications of the renal transplantation.
- Renal perfusion and functional studies using  $^{99m}\text{Tc}$ -DTPA and  $^{99m}\text{Tc}$ -MAG3 offer comprehensive evaluation of the renal blood flow, function and drainage.

### **Q13. Describe mammography technique and interpretation.**

#### **Mammographic evaluation of various patterns of breast lesions and BI-RADS staging system.**

**Ans.**

- Imaging is essential for accurate diagnosis of breast diseases and early detection of breast cancer.
- Mammography and ultrasound are basic first-line investigations for breast imaging. MRI is a valuable adjunctive diagnostic tool because of its high sensitivity for breast pathology.
- Mammography is the only screening modality, which has been proven to reduce mortality from breast cancer through early detection.

#### **Mammography**

##### *Basic physics, equipment and technique*

- Effective mammography requires constant high quality images with optimal contrast, high resolution and low radiation dose.
- Special X-ray tube with molybdenum or rhodium anodes is required to produce the low energy X-ray necessary to achieve high tissue contrast. Additional filter may be used to remove very low energy radiation as it will get absorbed in breast tissue and increase radiation dose without any significant effect on image quality. The most commonly used target-filter combination is molybdenum with 0.03 mm molybdenum filter.
- Good breast compression with radiolucent compression paddle is mandatory for mammography. Breast compression results in uniform thickness and, hence uniform exposure and density in most parts of breast. The overlapping tissue is separated and spread evenly which improves their visualization. Firm compression of breast closer to

film or image receptor reduces the geometric blur and magnification. Compression also stabilizes the breast tissue and hence reduces the movement blur. Mammography is not possible if compression is not possible. Such situations may include uncooperative women, presence of wound, recent surgery or tender breast.

#### *Radiation dose*

- Modern mammography examinations are safe and the mean glandular dose to the breast is approximately **2 mGy per exposure**.
- With present mammography techniques, there is little or no radiation-related risk to the women over 40 years of age.

#### **Mammography projections**

- The standard mammography projections consist of mediolateral oblique (MLO) and cranio-caudal (CC) views of each breast.
- These provide two orthogonal images for basic imaging evaluation of the breast.
- The MLO view is obtained with the tube angled at 45 degree to the horizontal, with compression applied obliquely across the chest wall, perpendicular the long-axis of pectoralis major muscle.
- The standard CC view is obtained with a vertical X-ray beam. Positioning is achieved by pulling the breast up and forward, away from the chest wall, with compression applied from above. The CC view demonstrates subareolar, medial and lateral portions of breast; however, tissue on posterolateral aspect of breast may not be demonstrated completely.

#### **Supplementary views**

- **Rolled views:** On the rolled views, the summation shadow would be separated into its normal fibroglandular components, whereas true mass will retain their shape and persist.
- **Magnification views:** This is particularly helpful for detailed evaluation of microcalcification and the margins of small mass lesions.
- **Spot compression view:** By additional compression, the breast tissue overlying a small lesion is displaced, allowing for better demonstration of its morphological features.
- **Extended CC view:** It is used for demonstration of most posterolateral aspect of breast tissue.
- **Valley view:** It is used for most posteromedial aspect of breast tissue.

#### **Normal mammographic anatomy**

- The breast contains 15–20 lobes, each drained by a lactiferous duct with an opening in nipple.
- The main duct branches repeatedly within the breast and the most distal branches of the duct system are called the terminal ducts.
- The terminal ducts consist of intralobular and extralobular portions. The intralobular portion, together with the acini, forms a lobule. The extralobular duct with lobule is termed as terminal ductular lobular unit (TDL unit).
- TDL unit is the site of origin of most malignant and benign breast diseases.
- Mammographic appearance of breast tissue depends on relative amount of fat and glandular tissue that is present. The young woman's breast contains a large amount of glandular tissue which appears as soft tissue density on the mammogram; in older

patient, when most of the breast glandular tissue has involuted, most of the breast tissue appears as fatty density.

- Normal lymphnodes are often seen in axilla and within the breast in upper outer quadrant. A normal lymphnode is an oval or lobulated dense mass with a radiolucent fatty hilum.
- The nipple is usually everted, and it should be seen in profile on at least one mammographic views that the retroareolar region can be visualized without its superimposition. The nipple can be inverted as normal variant; however, recent nipple inversion is of concern for a retroareolar mass.

### **Interpretation and reporting of mammograms**

- American college of Radiology (ACR) has devised the Breast Imaging Reporting and Data System (BI-RADS), a standardized method of describing the morphology of breast lesions and categorizing the findings in an unambiguous report.
- The first step in describing mammogram is to assess the parenchymal density as the diagnostic accuracy of mammogram decreases with increased density of breast. The ACR BI-RADS divides breast parenchyma into four major categories based on mammographic densities.
  - Breast tissue almost entirely fatty (dense glandular tissue occupying less than 25% of breast tissue).
  - Scattered fibroglandular tissue (dense glandular tissue occupying 25–50% of the breast tissue).
  - Heterogeneously dense parenchyma (dense glandular parenchyma occupying 50–75% of the breast tissue).
  - Extremely dense breast (dense glandular tissue occupying >75% of the breast tissue).
- The abnormal mammographic findings are then categorized into mass, calcification, architectural distortion and asymmetry.

### **ACR BI-RADS assessment categories**

The mammographic findings are categorized from BI-RADS 0 to BI-RADS 6, which are primarily aimed for communication of significance of mammographic findings to the referring physician and to recommend most appropriate management.

#### *Category 0: Incomplete evaluation*

- Additional views, repeat mammography or ultrasound or MRI are recommended.

#### *Category 1: Negative*

- There is nothing to comment on. The breasts are symmetric and no masses, architectural distortion or calcification noted.

#### *Category 2: Benign findings*

- Involuting calcified fibroadenoma, multiple secretory calcification, fat containing lesions such as oil cysts, lipomas, galactoceles and mixed density hamartomas all have characteristically benign appearances.
- The reader may also choose to describe intramammary lymph nodes, vascular calcification, implants or architectural distortion clearly related to the prior surgery while still concluding that there is no mammographic evidence of malignancy.

*Category 3: Probably benign findings—initial short interval follow-up is suggested*

- A finding put in this category should have less than 2 percent risk of malignancy.
- Lesions appropriately placed in this category include non-neoplastic, circumscribed mass on a baseline mammogram, a focal asymmetry and a cluster of punctate calcifications.

*Management*

- A short-term follow-up (normally 6 months later) mammogram of the same breast is performed for the stability of the lesion. The follow-up is aimed to prove the benign diagnosis short of biopsy and not to determine whether the lesion is benign or malignant.
- If the lesion is stable, the follow-up is continued usually for two years. Any change in morphology in follow-up mammogram requires an urgent biopsy.

*Category 4: Suspicious abnormality—Biopsy should be considered*

- This category is reserved for the lesions that do not have classic appearance of malignancy and the probability of malignancy is higher than category 3. Probability of malignancy in category 4 is high, ranging from 5–95%.
- By dividing the category 4 into subcategories as 4A, 4B and 4C, it is encouraged that relevant probabilities for malignancy be indicated within this category so the patient and her physician can make an informed decision on the ultimate course of action.
- Category 4A is used if the lesion is sampled, but with a very low probability of malignancy. A malignant pathology report is not expected and 6 month follow-up after a benign biopsy is appropriate. Examples of findings in this category include palpable, partially circumscribed mass with ultrasound features suggestive of fibroadenoma, a palpable complicated cyst or a breast abscess.
- Category 4B includes the lesions with an intermediate probability of malignancy. Biopsy is recommended and close radiopathological correlation is warranted.
- Category 4C includes findings of moderate concern, but not classic for malignancy. Examples of the lesions placed in this category are solid, irregular masses with ill-defined margins or recent cluster of fine pleomorphic calcification. A malignant result is expected. In case of a benign biopsy report, 6 month follow-up is mandatory to rule out a false negative report.

*Category 5: Highly suspicious malignancy—appropriate action should be taken*

- This category is used for the findings classic for breast cancer, with a >95% likelihood of malignancy.
- A speculated, irregular high density mass, a segmental or linear arrangement of fine linear calcifications or an irregular speculated mass with associated pleomorphic calcification are examples of lesions that should be placed in this category.

*Category 6: Known biopsy proven malignancy—appropriate action should be taken*

- This category is reserved for lesions identified on the imaging study with biopsy proof of malignancy prior to definitive therapy.

#### **Q14. State imaging approach in a patient with blunt abdominal trauma.**

**What is FAST? Discuss the role of FAST in evaluation of trauma patients.**

**Ans.** Prompt and accurate clinical assessment is essential in the initial evaluation of an abdominal trauma patient. Unfortunately, the clinical evaluation is often unreliable. Neurological

impairment due to traumatic event itself or concomitant factors such as intoxication or inebriation significantly limits the usefulness of the clinical examination. The most reliable signs in conscious patients are pain and tenderness with guarding. Twelve to 16% of patients with abdominal trauma present in a state of shock. After initial evaluation and resuscitation, subsequent management depends on hemodynamic stability of the patient.

**As per ACR guidelines/appropriateness criteria, the patients are divided into following categories:**

*Category A*

- This category includes hemodynamically unstable patients following clinically obvious abdominal trauma and with unresponsive profound hypotension need rapid clinical evaluation and immediate resuscitation with volume replacement.
- If such unstable patients do not respond to resuscitation, and if they have clinical evidence of abdominal injury, they should go immediately to the operating room without imaging.
- During resuscitative efforts if time and circumstances permit, conventional radiographs of the chest and abdomen are often obtained as part of trauma protocols. This may help identify a pneumothorax, pneumoperitoneum or significant bone injuries.
- Ultrasound performed by an experienced sonologist to check for intraperitoneal free fluid may quickly provide information that can support a decision to operate immediately, with the caveat that the false negative rate at least 15%. More detailed ultrasound to check for organ injury takes too long in this setting and suffers from poor sensitivity.
- There is now general agreement that routine diagnostic peritoneal lavage is obsolete because of its invasive nature /lack of specificity, and inability to predict the need for therapeutic surgery.

*Category B*

- This category includes hemodynamically stable patients, patients with mild to moderate responsive hypotension presenting to emergency room after blunt abdominal trauma, and unstable patients who stabilize after initial resuscitation.
- These patients typically have a history significant trauma and have at least moderate suspicion of intraabdominal injury based on clinical signs and symptoms. These patients should be evaluated by imaging.
- In patients with clinical evaluation suggesting a lesser index of suspicion for significant intraabdominal injury, chest and abdominal radiographs, hematocrit with blood chemistries and a urinalysis should be performed.
- If these tests are unremarkable in the setting of a reliable clinical abdominal exam, a period of clinical observation may all be that is needed.
- However, if a reliable abdominal exam cannot be performed or if a clinical evaluation suggests organ injury, hemoperitoneum or peritonitis, further imaging is needed. The need for initial radiographs may be obviated if the clinical evaluation merits a computed tomography.
- Ultrasound is not a good modality for further imaging because it is relatively much less sensitive than computed topography for liver and spleen injuries and highly insensitive for renal, pancreatic, mesenteric, gut, bladder and retroperitoneal injuries. If due to



circumstances, a negative ultrasound is the sole imaging modality used to triage a patient, for safety reasons it must be followed by a 12–24 hour period of in-hospital observation. A negative ultrasound alone may be adequate to release the patient from observation only in a separate subcategory of stable patients with trivial trauma, a low clinical index of suspicion, and no signs or symptoms of intraabdominal injury. Any positive findings on ultrasound would, however, warrant computed tomography.

- Computed tomography is an excellent modality for detecting solid organ and gut injuries together with even small amount of hemoperitoneum. The findings of computed tomography in combination with clinical status of the patient plays an important role in deciding whether a patient needs urgent therapeutic surgery or therapeutic angiography or whether can be managed conservatively. Identification of active hemorrhage, parenchymal blush or pseudoaneurysm in spleen, gut perforation, diaphragmatic injury and pancreatic injury tilt the scales towards surgical or angiographic management. Selected stable patients with negative computed tomography findings in which surgical intervention is not required may be managed conservatively under observation with imaging follow-up.

#### *Category C*

- This category includes patients with hematuria which require some modification to imaging work up.
- Patients with microscopic hematuria (<35 RBC per high power field) do not need specific urinary tract imaging.
- All patients with microscopic hematuria greater than 35 red blood cells per high power field, with macroscopic hematuria, or with fracture/diastasis of the symphysis pubis and its rami plus any hematuria need imaging of the urinary tract.
- If the urethral meatus has gross blood, if there is a floating prostate, or if a Foley catheter cannot be passed, a retrograde urethrogram should first be performed to rule out urethral injury. However, if clinical evaluation or urethrogram indicates no urethral injury, a computed tomography cystogram should be added to the abdominal computed tomography.

#### **FAST and eFAST**

Sonography is commonly used for the initial assessment of abdominal trauma. Abdominal ultrasound in cases of major trauma is usually performed with a FAST (Focused Assessment with Sonography in Trauma) examination. It provides a fast overview of abdomen to detect free fluid which indicates hemoperitoneum and visceral organ injury in this setting.

#### *Indication*

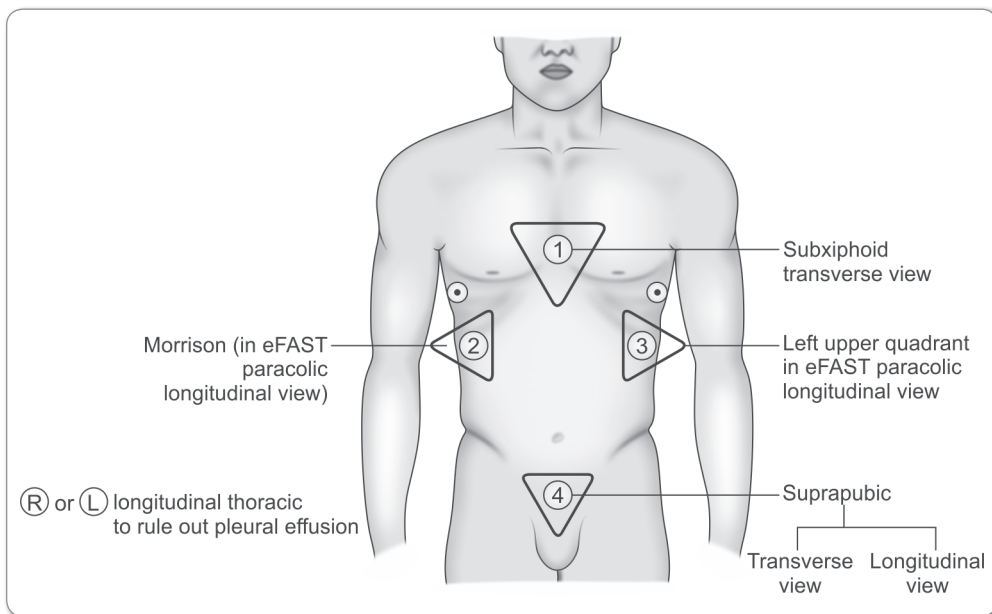
- The main use of US in patients with blunt or penetrating trauma is in screening for abdominal injuries.
- In hypotensive patients and those whose condition is unstable, US can help determine whether immediate surgery is needed before the patient undergoes a further evaluation with CT.
- The FAST scan should be completed within few minutes with an aim to primarily research for free intraperitoneal fluid and screens for organ injuries may be detected, search for such injuries should not delay the examination especially in the setting suspicion of hemorrhage.

- According to ACR appropriateness criteria, in hemodynamically unstable patients, bedside ultrasound to check for intraperitoneal free fluid may quickly provide information that can support a decision to operate immediately, with the caveat that the false negative rate is at least 15%. Hemodynamically stable patients with clinical or laboratory evidence of major abdominal injuries should be evaluated by CECT, however, ultrasound can be used as a primary imaging modality for screening if availability of CT is limited.

#### Technique

- For a FAST examination, the patient is placed supine, if possible. Use of a mobile US machine is recommended because standard placement of the patient is not always possible.
- The depth of ultrasound wave penetration for abdominal US must be at least 20 cm, which usually requires the use of a 3.5–5.0 MHz convex transducer.

The following four standard views should be obtained:



**Fig. 1:** FAST probe positioning

1. Transverse view of the subxiphoid region to diagnose pericardial effusion and injuries to the left lobe of the liver.
2. Longitudinal view of the right upper quadrant to show the right lobe of the liver, the right kidney, and the space between the two (the Morison pouch), which may fill with peritoneal fluid when the patient is supine.
3. Longitudinal view of the left upper quadrant to show the left kidney, the spleen, and the space between them, which also may contain free intraperitoneal fluid.
4. Transverse and longitudinal views of the suprapubic region to depict the urinary bladder and rectouterine or retrovesical pouch, a recess formed by a fold of the peritoneum that descends between the rectum and uterus in women or the rectum

and bladder in men. This recess is called the pouch of Douglas. Like the Morison pouch, it is a space in which free intraperitoneal fluid may collect.

In addition to these four standard views, a right and a left longitudinal thoracic view may be acquired to rule out pleural effusion. Because these views can be obtained quickly, they should be included in routine FAST acquisitions in all patients with trauma to the chest. Longitudinal views of bilateral flanks should be obtained to look for bilateral paracolic gutters. When these additional views are also included, the study is labeled as extended FAST (eFAST).

- When the FAST examination is performed correctly by an experienced sonographer, it ordinarily takes no more than 5 minutes. However, in some cases, it may be difficult to obtain the standard views, and the examination then will be prolonged. The operator should not waste too much time with the FAST examination if there is any suspicion of hemorrhage.

### **Ultrasound appearance of free fluid**

- In a technically successful examination, hemoperitoneum is visualized as a lenticular collection in the subphrenic space, triangular in Morison's pouch and ovoid in pelvis.
- Solid organ injury can be recognized by subcapsular or intraparenchymal hematoma. US appearance of hematoma depends on multiple factors, especially on its duration.
- The early hematoma is echogenic but gradually progresses to sonolucency over 96 hours.

### **Sensitivity and specificity**

- The reported sensitivity of FAST for detection of free intraperitoneal fluid ranges from 0.64–0.98 and specificity from 0.86–1.00. False negative rate of FAST is average 15% for fluid detection. Negative FAST should be indicating the absence of hemoperitoneum, not the absence of intraabdominal injury. However, it should be remembered that the sensitivity of FAST for free fluid is less than CT. A negative FAST should be viewed with suspicion if the finding is not commensurate with patients' clinical presentation.
- A positive FAST does not necessarily mandate laparotomy. Hemodynamically stable patients with positive FAST should have their injuries staged with CT, giving them the benefit of non-operative management whenever possible.
- FAST is not reliable for assessment of retroperitoneum.
- The sensitivity of ultrasound for detection of liver injury ranges from 0.15–0.88 and it misses average 15% injuries to liver. The sensitivity for splenic injuries is reported to be between 0.37–0.85 with average false negative rate of more than 50%. Sensitivity for renal and pancreatic injuries is less than of liver and spleen and even lesser for bowel and mesenteric injuries.
- The sensitivity for pericardial effusion is high (0.97–1.00).

### **Advantages**

- US has obvious advantages in that it is widely available, easy to perform, and low cost.

### **Limitations**

- Operator dependent.
- Limited by excessive bowel gas (especially in post-traumatic paralytic ileus).
- Limited by open wounds and bandages and by cutaneous emphysema.

**Q15. Discuss radiological features of acute intestinal obstruction.****Role of radiological imaging in evaluation of acute intestinal obstruction.****Imaging approach in patient with acute intestinal obstruction.**

**Ans.** Intestinal obstruction is a common clinical abnormality that is usually suspected on the basis of clinical signs and patient history. However, it is essential to manage treatment properly, to determine the site, level, and cause of obstruction, and even to try to establish a prognosis prior to surgery.

**Diagnostic modalities:**

- Plain radiograph: Supine and standing AP (at least 10–15 minutes of standing position)
- Ultrasound: Limited role especially when bowel loops are filled with gas.
- Computed tomography: Contrast enhanced CT without oral contrast. Owing to the capability of CT for early demonstration of strangulation, CT is now considered the best modality for determining which patients would benefit from conservative management and close follow-up and which patients would benefit from immediate surgical intervention.

The diagnosis of intestinal obstruction may be done alone on basis of plain film findings. Additional imaging with ultrasound and CT is required in equivocal cases to confirm the diagnosis, the level and cause of obstruction.

Nowadays, owing to the increased application of advanced modalities of abdominal imaging in the clinical context of SBO, combined with the widespread assumption that most of these conditions resolve spontaneously with nonsurgical treatment, namely nasointestinal decompression, imaging has become the primary focus in the treatment of patients with SBO.

**Findings on plain radiography**

- The key radiographic signs that allow distinction between a high grade SBO and a low grade obstruction are the presence of small bowel distention, with maximal dilated loops averaging 36 mm in diameter and exceeding 50% of the caliber of the largest visible colon loop as well as a 2.5 times increase in the number of distended loops in the abdomen compared with the normal number.
- Other findings that are most significant and predictive of high-grade SBO, according to experienced gastrointestinal radiologists, are the presence of more than two air-fluid levels, air-fluid levels wider than 2.5 cm, and air-fluid levels differing more than 2 cm in height from one another within the same small bowel loop.
- Diagnosis of both small and large bowel loops dilatation till rectum is paralytic ileus. It may not be always possible to differentiate mechanical obstruction from ileus on frontal views. Lateral radiograph may help in this condition by showing air in rectum; however, ultrasound or contrast study may be required in equivocal cases.
- Distinction between small and large bowel loops on plain radiograph is carried out by central position of small bowel loop, presence of valvulae conniventes in jejunum and feature less tubular appearance of ileum.
- At times, the bowel loops proximal to the site of obstruction are completely filled with fluid resulting in appearance of gasless abdomen. When tiny amount of gas accompanies large amount of intraluminal fluid, small gas bubbles get entrapped between valvulae

conniventes resulting in the “string of beads” appearance. The appearance is virtually diagnostic of mechanical obstruction and almost never seen in paralytic ileus.

- Presence of thumb printing due to submucosal edema or hemorrhage should suggest ischemia in bowel loops and strangulated obstruction.
- Presence of intramural gas in the form of parallel streaks of gas along the bowel wall or as rings may also be seen in infants with necrotizing enterocolitis. These appearance should not be confused with bubbly appearance of pneumocystis coli which is a benign condition affecting the colon in adults.
- The cause of obstruction is different in different age groups. Duodenal atresia shows typical double bubble sign. In young children, intussusception causes mass-like soft tissue shadow with crescent of gas surrounding it.
- Sigmoid volvulus is generally seen in old age. The distended ahaustral sigmoid loop assumes an inverted U-shape with its apex under the dome. The opposed inner walls of the loops appear as a single line giving coffee bean appearance. Cecal volvulus is generally seen in young patients and shows haustra.

### **Findings on ultrasound**

- Despite being an operator-dependent technique and having inherent limitations in the evaluation of gas-containing structures, abdominal sonography can be quite valuable in certain situations, with high sensitivity in demonstrating the presence of SBO, its level, and in some instances the cause and severity of the obstruction.
- At sonography, bowel obstruction is considered to be present when the lumen of the fluid-filled small bowel loops is dilated to more than 3 cm, the length of the segment is more than 10 cm, and peristalsis of the dilated segment is increased, as shown by the to-and-fro or whirling motion of the bowel contents . The level of the obstruction is determined by means of the location of the bowel loops and the pattern of the valvulae conniventes.
- As with other cross-sectional imaging techniques, the cause of the SBO may be determined by examining the area of transition from the dilated to normal bowel.
- Causes of SBO like bezoars, intussusception, Crohn disease, and tumors can be depicted with this method. Obstruction associated with external hernias is ideal for sonographic detection in that dilated loops of intestine may be traced to a portion of the gut with normal caliber but abnormal position.
- The severity of the obstruction can also be assessed. The presence of free fluid between dilated small bowel loops, aperistalsis, and wall thickening (>3 mm) in a fluid-filled distended bowel segment suggests bowel infarction.

### **Findings at multidetector CT**

Results of multidetector CT can provide answers to specific questions that have a major effect on the clinical treatment of the patient. These questions include the following:

- Is the small bowel obstructed?
- What is the grade of severity of the obstruction?
- Where is the transition point?
- What is the cause of the obstruction?
- Are there any associated complications?

**Is the small bowel obstructed?**

- CT criteria for SBO are the presence of dilated small bowel loops (diameter >2.5 cm from outer wall to outer wall) proximally to normal-caliber or collapsed loops distally.
- When CT findings are equivocal for the presence of obstruction after positive oral contrast material has been given, it is often helpful to perform delayed scanning to assess the passage of contrast material. A complete obstruction is considered to be present when there is no passage of contrast medium beyond the point of obstruction on delayed scans obtained at 3–24 hours.
- A low-grade partial SBO is considered present when there is sufficient flow of contrast material through the point of obstruction. High-grade partial SBO is diagnosed when there is some stasis and delay in the passage of the contrast medium, so that diluted oral contrast material appears in the distended proximal bowel and minimal contrast material appears in the collapsed distal loops.

**What is the grade of severity of the obstruction?**

- In a high-grade obstruction, there is a 50% difference in caliber between the proximal dilated bowel and the distal collapsed bowel.
- A high-grade obstruction that has been present for several days leads to complete evacuation of the contents of the bowel segments distal to the obstruction point, highlighting the discrepancy in caliber between the proximal and distal small bowel loops.
- The small bowel feces sign is present when intraluminal particulate material is identified in the dilated small bowel. Its prevalence is low (7–8%), and it is described by some authors as being more likely to occur in moderate and high-grade obstruction.

**Where is the transition point?**

- The transition point is determined by identifying a caliber change between the dilated proximal and collapsed distal small bowel loops.

**What is the cause of obstruction?**

- A rule of thumb never to forget is that the answer is almost always in the transition point. Most intrinsic bowel lesions are seen at the transition point and manifest as localized mural thickening. Most extrinsic causes are seen adjacent to the transition point and usually have associated extraintestinal manifestations.

Causes of intestinal obstruction can be intrinsic to the bowel wall or lumen and extramural or extrinsic as follows:

**Intrinsic**

- **Intraluminal**
  - Foreign bodies [coin, battery], food or fecal impaction, worm ball [ascaris], bezoars, gallstones.
- **Intramural**
  - Stricture, malignancy.

**Extrinsic or extramural**

- Adhesions, hernia, malignancy, volvulus, intussusception.

**Is the obstruction simple or complicated?**

- On the basis of the pathophysiology of the obstructive process in the small bowel, SBO can be divided into two types: Simple obstructions and closed-loop obstructions.
- Simple obstruction of the bowel is considered when the bowel is occluded at one or several points along its course. The proximal part of the bowel is variably distended, depending on the severity and duration of the process.
- Closed-loop obstructions are diagnosed when a bowel loop of variable length is occluded at two adjacent points along its course. The occlusion can be partial or complete.
- The configuration of closed loop obstruction can be U-shaped or C-shaped, depending on the orientation of the closed loop. Because of the presence of constrictions of two adjacent bowel segments and the intervening mesentery, a narrow pedicle can be formed, leading to torsion of the loops and producing a small bowel volvulus.
- At CT, a “beak sign” is seen at the site of the torsion as a fusiform tapering, and occasionally a “whirl sign” can be seen, reflecting rotation of the bowel loops around the fixed point of obstruction.
- Strangulation is defined as a closed-loop obstruction associated with intestinal ischemia. This condition is seen in approximately 10% of patients with SBO, mainly when there is a delay in establishing the correct diagnosis and subsequent surgical treatment. It is associated with a high mortality rate. Findings indicative of strangulation include thickening and increased attenuation of the affected bowel wall, a halo or “target sign,” pneumatosis intestinalis, and gas in the portal vein, but these findings are not specific for strangulation. A specific finding is lack of wall enhancement; asymmetric enhancement or even delayed enhancement may also be found. Localized fluid and hemorrhage in the mesentery can also be seen.

**Q16. Discuss the role of barium swallow in esophageal disorders.**

**What is the differential diagnosis of dysphagia? Describe the role of barium swallow in evaluation of dysphagia.**

**Ans.**

**Dysphagia**

Subjective awareness of swallowing difficulty during passage of solid or liquid bolus from mouth to the stomach.

**Odynophagia**

Defined as painful deglutition.

**Causes of dysphagia***Mechanical dysphagia*

- Luminal
  - Large bolus
  - Foreign body
- Intrinsic narrowing
  - Inflammatory conditions causing edema and swelling
    - Stomatitis
    - Pharyngitis

- Epiglottitis
- Esophagitis
- Webs and rings
  - Pharyngeal (Plummer Vinson syndrome)
  - Esophageal (congenital, inflammatory)
  - Lower esophageal (Schatzki's ring)
- Benign strictures
  - Peptic
  - Caustic and drug induced
  - Inflammatory
  - Ischemic
  - Postoperative
  - Post radiation
  - Congenital
- Benign tumors
  - Lipoma
  - Leiomyoma
  - Angioma
  - Epithelial papilloma
  - Inflammatory fibroid polyp
- Malignant tumors
  - SCC
  - Adenocarcinoma
  - Lymphoma
  - Carcinosarcomas
  - Pseudosarcoma
  - Melanoma
  - Kaposi's sarcoma
- Extrinsic compression
  - Cervical spondylitis
  - Vertebral osteophytes
  - Retropharyngeal abscess or tumor
  - Enlarged thyroid gland
  - Zenker's diverticulum
  - Vascular compression
  - Posterior mediastinal mass
  - Post vagotomy hematoma or fibrosis

#### *Motor dysphagia*

- Difficulties in initiating swallowing reflex
  - Paralysis of tongue
  - Lack of saliva (Sjogren syndrome)
  - Sensory component lesion of 9th, 10th nerve



- Lesions of swallowing center
- Oropharyngeal anesthesia
- Disorders of pharyngeal and esophageal striated muscle
  - Muscle weakness
    - Lower motor neuron lesions: MND, polyneuritis, familial dystonias, CVA
    - Neuromuscular lesions: MG
    - Muscular disorders: Polio, dermatomyositis, myopathies
  - Nonperistaltic contractions or impaired inhibition involving
    - Pharynx and upper esophagus: Rabies, tetanus, UMN
    - UES: Palsy of suprahyoid muscles, cricopharyngeal achalasia
- Disorders of smooth muscles
  - Paralysis of esophageal body causing weak contractions
    - Scleroderma and related collagen vascular diseases
    - Hollow visceral myopathies
    - Metabolic neuromyopathy
    - Achalasia (classical)
  - Nonperistaltic contractions or impaired inhibition involving
    - Esophageal body: DES, vigorous achalasia
    - LES
      - **Achalasia:** Primary
      - **Secondary achalasia:** Chagas disease, carcinoma, lymphoma, toxins and drugs.

#### Techniques of examination

- **Chest X-ray:** It can reveal mediastinal widening, air fluid levels, foreign body
- **Barium swallow**
  - Single contrast for motility disorders and gross mass lesions or extrinsic compression and double contrast for mucosal detail examination.
  - **Contraindication:** Suspicion of aspiration or leakage into mediastinum.
- **Endoscopic ultrasound**
  - Advantage is high resolution EUS that shows layers of the esophageal wall that cannot be resolved on CT and MRI and, therefore, better for local tumor staging (best for differentiating T1 and T2 stage tumors).
- **CT**
  - Information about local spread of disease and involvement of adjacent structures.
  - Evaluation of abdominal structures for metastasis.
- **MRI**
  - Excellent details of spatial relationships of esophagus and especially pharynx.
- **Endoscopy:** Details of mucosa
  - To take biopsy.
  - therapeutic interventions.
- **Manometry:** Motility disorders.
- **pH monitoring:** Gold standard for gastroesophageal reflux.
- **Radionuclide imaging:** To assess gastroesophageal reflux.

**Structural diseases of pharynx***Foreign body*

May be located in vallecula, pyriform sinus or more commonly above cricopharyngeus muscle. A mucosal tear may cause considerable pain, i.e. odynophagia or dysphagia.

*Latertal pharyngeal outpouching*

- Also known as **hypopharyngeal ears**.
- Caused by defective functioning of constrictors which may cause dysphagia.
- Appears as large smooth marginated barium and air-filled bulges, located between hyoid bone and thyroid cartilage.
- Found in elderly patients and glass blowers.

*Zenker's diverticulum*

- Located in midline and protrude to left arising from Killian's dehiscence.
- Age of presentation: 60–70 yrs.
- Measure usually 2–6 cm in size.
- Symptoms: Retained food, regurgitation several hours after meal, dysphagia or aspiration pneumonia.
- Found in 2% of nonspecific dysphagia.
- Seen as contrast filled outpouching in lower cervical region traversing laterally to left.

*Killian jamieson's diverticulum*

- Rare entity.
- Located inferior to cricopharyngeus muscle and protrude laterally.
- Smaller than Zenker's diverticulum.
- May be bilateral.

*Webs*

- Thick (1 to 2 mm) diaphragm-like membrane that extends partially or completely around the lumen.
- Common cause of dysphagia.
- Sex predilection: F>M.
- Typically located at the anterior wall of hypopharynx: Esophageal junction at level of cricopharyngeus muscle.
- Appears 1 mm thick, located anteriorly.
- Associated with Plummer Vinson syndrome (iron deficiency anemia, pharyngeal atrophy, glossitis).
- Usually asymptomatic, can cause dysphagia for solids.

**D/D-** submucous venous plexus

**Esophageal motility disorders**

It can be divided into primary and secondary types.

- **Primary:** Affecting esophagus primarily.
- **Secondary:** Affecting esophagus secondarily to other systemic involvement.

**Primary motility disorders***Achalasia*

- **Manometric studies** reveal:
  - Absent peristalsis
  - Incomplete LES relaxation
  - Non-propulsive contraction
  - Increased LES pressure
  - Positive response to amyl nitrate.

**Radiographically:***On plain chest radiographs*

- *A double mediastinal stripe* is occasionally depicted.
- *An air-fluid level* can be seen in the esophagus; that is frequently retrocardiac.
- *The gastric air bubble may be small or absent.*

*On barium study*

- *Failure of peristalsis to clear the esophagus of barium* with the patient in the recumbent position.
- *Antirade and retrograde motion of barium* in the esophagus secondary to tertiary contractions.
- *LES relaxation that is incomplete* and not coordinated with esophageal contraction.
- *Dilation of the esophageal body*, which is maximal in the distal esophagus.
- *Pooling or stasis of barium in the esophagus* when the esophagus has become atonic or noncontractile.
- Tapering of the barium column at the unrelaxed LES, resulting in **the bird beak sign** (not a specific sign for achalasia, seen in conditions in which lower esophageal sphincter fail to relax like presbyesophagus, DES, connective tissue diseases with LES relaxation failure, carcinoma esophagus).

*Achalasia variants:*

- **Vigorous achalasia:** Shows simultaneous repetitive contractions of high amplitude (>60 mm Hg). Patients present with chest pain & have less esophageal dilatation.
- **Early achalasia:** Shows aperistalsis but LES relaxation. Radiographic findings are similar except for less esophageal dilatation.

**Pseudoachalasia**

- Pseudoachalasia is a term used to describe the clinical picture of gastroesophageal junction obstruction presenting with the radiologic diagnosis of achalasia.
- In such patients, manometric findings are inconsistent with those in achalasia.

*Causes include:*

- Esophageal malignancy.
- Gastric malignancy.
- Chagas disease.

- Post vagotomy.
- Collagen vascular disorders.
- Lymphoma.

Amyl nitrite (smooth muscle relaxant) test distinguishes achalasia from pseudo achalasia. Patients with achalasia will show considerable increase in diameter of lower esophageal sphincter (>3 mm increase) while patients with secondary achalasia fail to do so.

### **Diffuse esophageal spasm**

- Primary peristalsis present in upper esophagus but intermittently absent in smooth muscle segment.
- Non-peristaltic contractions which are repetitive, simultaneous, and may obliterate esophageal lumen causing “cork-screw or rosary bead” appearance.
- Compartmentalization of barium.
- Shish kebab appearance.
- Esophagus is not dilated.

### **Hypertensive peristalsis**

It also known as *nut cracker esophagus*

- **Manometry** reveals greater than 200 mm Hg pressure rise in smooth muscle part of esophagus (normal <40 mm Hg).
- **At fluoroscopy:** Peristalsis usually appears normal hence manometric correlation is valuable.

### **Structural lesions of esophagus**

*Reflux (peptic) esophagitis*

- Earliest changes seen at endoscopy: The mucosa become red and edema results in loss of vessel pattern, blurring of squamocolumnar junction.
- Only with more pronounced edema changes of a fine mucosal modularity is seen on a DC barium swallow.
- The collapsed esophagus shows thickened longitudinal folds (wider than 3 mm), which when nodular, give appearance of varices.
- Local muscular spasm may produce transverse folds. Scarring produces permanent folds that radiate from the margins of ulcers with barium trapped between the folds, producing a characteristic “**Step-Ladder**” appearance.
- When viewed in profile outpouching between folds can mimic ulceration, but unlike ulcers they change shape during the examination.
- These coarse transverse folds are easily differentiated from the transient fine mucosal folds which are thought to result from contraction of the muscularis mucosa (feline esophagus), often seen when the esophagus is only partially distended.
- These fixed transverse folds tend to be wider than those in the feline esophagus and usually do not extend more than halfway across the esophagus.
- Severe scarring results in stricture formation.
- Mild strictures may be difficult to identify at endoscopy but often demonstrated at barium swallow, particularly bread barium. This gives useful information about localized non- distensibility or areas of poor contraction.

- A peptic ulcer above a hiatus hernia is typically short, and has a smooth lumen and tapered margins, may be mistaken for schatzki rings. However, asymmetric scarring and ulceration may produce a stricture with irregular margins similar to that of carcinoma.
- Long peptic stricture may be seen in Zollinger-Ellison syndrome.
- With further progression, multiple fine ulcers produce a punctate or granular appearance or large discrete punched-out ulcers develop. Ulceration is more pronounced immediately above the EGJ.

### **Esophageal diverticula**

Most of oesophageal diverticula are acquired and type of pseudo diverticula.

#### *Classification*

- Pulsion diverticula of upper part (zenker's diverticula)
- Traction diverticula of middle part- small number of midesophageal diverticula, conical in shape and having no neck.
- Pulsion diverticula of lower part (epiphrenic)- lies above LES

#### *Barium study*

- Small (0.5-2 cm) diverticulae are seen as transient outpouchings.
- Large diverticulae appear as persistent outpouching which empty only by gravity and may compress adjacent oesophagus

### **Foreign bodies**

#### *Plain film*

- Usually oriented in coronal plane. Gas fluid level/tracheal displacement may have seen.

#### *Contrast examination*

- Water soluble, non-ionic contrast is used
- Cotton balls/ marshmallow soaked in barium may be used.

### **Diaphragmatic hernia**

#### **Sliding hiatus hernias**

##### *Barium study:*

- Patient lies in LPO position and drinks Barium through a straw.
- Spot films of lower esophagus and gastro-esophageal junction are taken, when they distend when barium passes through them.
- Three or more gastric folds may be identified passing from the stomach across the hiatus and the Z- line and areae gastricae are located above the hiatus.
- However, barium studies do not normally define the mucosal junction and only rarely Z line is identified.
- In this circumstance, the diagnosis rests on demonstration of secondary features which indicate the position of the mucosal junction.
  - **Schatzki's or B ring:** if present, is pathognomonic of a hiatal hernia because it represents Z line and marks squamocolumnar junction.
  - **Hiatal width:** the diameter of maximally distended hiatal segment is equal to or more than the two thirds of that of the maximally distended thoracic segment, i.e. About 2.5 cm or more.

*CT scan*

- On CT the diaphragmatic crura are separated by  $> 15$  mm, and the hernia produces a mass of soft-tissue density that protrudes above the hiatus and which may be surrounded by mesenteric fat.

**Rolling hiatus hernia**

- A rare type of diaphragmatic hernia in which the cardia remains below the diaphragm while the fundus herniates through a weakness or tear in the phrenico-esophageal membrane to lie alongside the lower esophagus
- Only slight predisposition to reflux
- Common complication is anemia from chronic bleeding
- Most paraesophageal hernias are not reducible
- When both sliding and paraesophageal component is present, it is called mixed hiatus hernia.

**Esophageal webs**

Thick (1 to 2 mm) diaphragm like membrane that extend partially or completely around the esophageal lumen.

*Types*

- Idiopathic
- Associated with Plummer Vinson syndrome
- Due to Epidermolysis bullosa or GVH reaction
  - Histology-Plication of normal squamous mucosa with inflammation
  - MC site is within few cms of cricopharyngeus
  - May represent heterotrophic mucosa (gastric)
  - Radiologically it is located in proximal oesophagus, anteriorly. Commonly seen on lateral view and can cause jet effect.

**Oesophageal rings**

Short annular narrowing 4 to 10 mm in vertical extent.

- MC ring is Schatzki's ring -asymptomatic mucosal narrowing at GE junction.
- Symptomatic schatzki's ring is 4-5 mm with narrowing of luminal aperture.
- Symptoms—streak house syndrome
  - Sub sternal pain
  - Episodic dysphagia for solids
  - Obstruction may be relieved by drinking or regurgitation.

**On fluoroscopy** appear as thickened annular narrowing at GE junction

- If lumen  $< 12$  mm ----always symptomatic
- Hiatus hernias generally associated
- Marshmallow or barium tablet may be helpful in demonstration.

**Esophageal tumors***Malignant*

- Carcinoma—SCC, adenocarcinoma, carcinoid

- Sarcomas—fibrosarcoma, leiomyosarcoma, lymphosarcoma, others
- Metastasis

#### *Benign*

- Mucosal—papilloma, adenoma
- Sub mucosal—fibroma, lipoma, hemangioma, leiomyoma, neurofibroma

#### *Non-malignant*

- Fibro vascular polyp
- Cystic lesions—retention, enteric, duplication cyst
- Solitary varix
- Ectopic tissue
- Hematoma
- Hamartoma

#### **Indentations**

- Normal impressions
  - Aortic knob
  - Left bronchus
  - Diaphragmatic hiatus.
- Common in elderly
  - Ectatic arch
  - Ascending aorta
  - Cervical nodes.
- Heart
  - Enlarged left atrium
  - Generalized cardiomegaly
  - Cardiac tumor.
- Aortic arch anomalies
  - Right sided aortic arch
  - Cervical arch
  - Double aortic arch
  - Aortic coarctation
  - Aortic aneurysm.
- Other blood vessels
  - Aberrant right subclavian artery
  - Anomalous left pulmonary artery
  - Dilated bronchial artery
  - Left inferior pulmonary vein
  - Common pulmonary vein
  - Dilated hemiazygous vein.
- Mediastinal masses
  - Enlarged nodes
  - Mesenchymal tumors

- Hematoma
  - Abscess.
- Cervical masses
  - Cervical adenopathy
  - Thyroid tumor
  - Thyroid goiter
  - Parathyroid tumor.



# SECTION

# 8

## Miscellaneous Topics

- Anesthesia
- Skin
- Burns and Plastic Surgery
- Head and Neck Surgery
- Oncosurgery Basics



## ANESTHESIA

### Q1. Write a note on pre-anesthetic medication.

**Ans.** Preanesthetic medication is the use of drugs to improve the intraoperative status of patient and the postoperative recovery from anesthesia.

#### **Aims of preanesthetic medication**

- Decrease anxiety and produce sedation
- Improve postoperative analgesia
- Decrease secretions
- Improve amnesia
- Decrease PONV (Postoperative nausea and vomiting)
- Decrease acid secretion and postoperative eructations and acidity
- Decrease chances of aspiration due to its role on decreasing secretions and antiemetic effect as well as decrease the injury from aspiration by decreasing the acidity
- Preanesthetic drugs for the patients with specific disorders: Pheochromocytoma, patients with steroid dependence, patients with epilepsy or arrhythmias, etc.

#### **Drugs commonly used as preanesthetic medication**

- Alprazolam, midazolam or diazepam on the night before surgery or same day at early morning
- Morphine/Voveran and related drugs for analgesia 1 hour before surgery
- Pantoprazole or Ranitidine for antacid effect about 1 hour before surgery
- Metoclopramide or ondansetron or palonosetron 1 hour before surgery
- Atropine or other anticholinergic drugs to decrease secretions, decrease laryngospasm, decrease choking, and decrease cardiac vagal stimulation and cardiac arrest
- Barbiturates and neuroleptics like chlorpromazine are also used as preanesthetic medication.

### Q2. Describe the ASA grade of the patients planned for surgery.

**Ans.** ASA status (American Society of Anesthesiologists physical status score) is for the physical status of the patients which also give a guide to the choice of anesthetic of the patients as well as record of patient's preoperative health status for postoperative comparison or statistical analysis and is as follows:

#### **ASA 1**

A normal healthy patient. No organic, physiologic, or psychiatric disturbance. A healthy patient with good exercise tolerance.

#### **ASA 2**

A patient with mild systemic disease which is well controlled, e.g. controlled hypertension or diabetes without systemic effects, mild obesity, pregnancy.

#### **ASA 3**

A patient with severe systemic disease. Has a controlled disease of more than one body system or one major system but no immediate danger of death; Previous myocardial

infarction, stable angina, controlled congestive heart failure (CHF), poorly controlled hypertension, morbid obesity, chronic renal failure.

#### ASA 4

A patient with severe systemic disease that is a constant threat to life, e.g. unstable angina, symptomatic congestive heart failure or COPD.

#### ASA 5

A moribund patient who is not expected to survive without the operation for > 24 hours, e.g. multisystem failure, hemodynamic instability.

#### ASA 6

A brain-dead patient whose organs are being removed for donation.

#### ASA 'E'

'E' is applied to ASA class when the patient requires emergency surgery, e.g. "ASA 3E" and so on. ASA 6E does not exist (Obvious!!!) and almost all ASA 5 cases are actually ASA 5E.

### Q3. Write a note on muscle relaxants.

**Write a note on Mivacurium.**

**Write a note on succinylcholine.**

Ans.

#### Classification of muscle relaxants

Centrally acting	Mephenesin group		Mephenesin Carisoprodol Chloroxazone Chlormezanone Methocarbamol
	Benzodiazepine congener		Diazepam
	Central Alpha-2 agonist		Tizanidine
	GABA derivative		Baclofen
Peripherally acting	Directly acting		Dantrolene Quinine
	Neuromuscular blocking	Depolarizing (Noncompetitive)	Succinylcholine Decamethonium
		Nondepolarizing (Competitive)	<b>Short acting</b> Mivacurium  <b>Intermediate acting</b> Atracurium, Cisatracurium, Rocuronium, Vecuronium  <b>Long acting</b> Doxacurium, Pancuronium, Pipecuronium, D-Tubocurarine

#### Mechanism of action

- **Nondepolarizing blockers:** On endplate of skeletal muscle, the drugs compete with acetylcholine for neuromuscular blockers and decrease the frequency of calcium channel

opening. They also block prejunctional nicotine receptors on motor endplate endings and exhibit fade phenomenon. Neostigmine antagonises its actions.

- **Depolarizing blockers:** These drugs have affinity and intrinsic activity. Phase 1 block produces rapid, persistent depolarization which is not antagonized by neostigmine and phase 2 block produces slow depolarization which is partially reversed by neostigmine.
- **Centrally acting muscle relaxants:** Act on cerebrospinal axis without altering consciousness, act on spinal and supraspinal polysynaptic reflexes which are involved in regulation of muscle tone without affecting monosynaptic stretch reflex. However, they can produce sedation due to action on ascending reticular activating system. They decrease tone but do not decrease power.

**GABA B agonists:** Increase potassium channel conductance and inhibit both monosynaptic and polysynaptic reflexes in the spinal cord.

#### Important points about muscle relaxants

- Shortest, fastest acting and least potent agent—Succinylcholine
- Longest acting—Pancuronium
- Shortest acting competitive agent—Mivacurium
- Fastest acting, least potent competitive agent—Rocuronium
- Most potent agent—Doxacurium
- D-Tubocurarine produces histamine release syndromes whereas succinylcholine produces vagal stimulation and ganglion stimulation
- Pancuronium, Pipecuronium, D-Tubocurarine and Doxacurium are metabolized in kidney whereas Vecuronium, Atracurium and Mivacurium are metabolized in liver. Atracurium and Cis-Atracurium are also eliminated by Hoffman elimination. Mivacurium is also metabolized by pseudocholinesterase—activity is measured using dibucaine number (Other drugs include succinylcholine, mivacurium, cocaine, procaine, Remifentanyl and Bambuterol)
- Succinylcholine and halothane can cause malignant hyperthermia in patients with pseudocholinesterase deficiency
- The drug of choice for treatment of malignant hyperthermia is Dantrolene. Procaine and Propofol can be used in the patients who have had malignant hyperthermia or pseudocholinesterase deficiency. Dantrolene is also the drug of choice in neuroleptic malignant syndrome
- Mephensin can cause hemolytic jaundice.

#### Uses

- Centrally acting muscle relaxants are used in treatment of amyotrophic lateral sclerosis, flexor spasms, spinal injuries and multiple sclerosis and tetanus to decrease spasticity
- Dantrolene is the drug of choice in malignant hyperthermia, neuroleptic malignant syndrome and patients with paraplegia, cerebral palsy, and upper motor neuron disorders
- Succinylcholine is used for endotracheal intubation as well as muscle relaxant before and during anesthetic procedures
- Tizanidine inhibits the release of excitatory amino acids in spinal interneurons and facilitated glycine. It is used in patients with spinal injury, stroke and multiple sclerosis.

**Collection of points for MIVACURIUM**

- It is a peripherally acting nondepolarizing (competitive) neuromuscular blocking muscle relaxant.
- **Mechanism of action**
  - On endplate of skeletal muscle, the drug competes with acetylcholine for neuromuscular blockers and decrease the frequency of calcium channel opening. They also block prejunctional nicotine receptors on motor endplate endings and exhibit fade phenomenon. Neostigmine antagonizes its actions.
  - Increase in dose increases the rapidity of onset of neuromuscular blockade.
- Mivacurium is the shortest acting competitive muscle relaxant
- It is metabolized in liver as well as by pseudocholinesterase (plasma cholinesterase). The activity can be measured by dibucaine number
- Mivacurium has a tendency for histamine release and therefore can reduce arterial pressure and systemic vascular resistance when used in clinical setting
- It is the drug of choice for day care surgery as well as for producing short duration muscle relaxation in anesthetic procedures.

**Collection of points for SUCCINYLCHOLINE**

- It is a peripherally acting depolarizing (noncompetitive) neuromuscular blocking muscle relaxant.

**Mechanism of action**

- These drugs have affinity and intrinsic activity. Phase 1 block produces rapid, persistent depolarization which is not antagonized by neostigmine and phase 2 block produces slow depolarization which is partially reversed by neostigmine
- It is the shortest, fastest acting and least potent agent
- It produces vagal stimulation and ganglion stimulation
- It is the most commonly used muscle relaxant to pass endotracheal tube.

**Contraindications**

- Burns, crush injury, rhabdomyolysis, muscular dystrophy, Guillain barre syndrome, myasthenia, paraplegia and hemiplegia as in these patients, it can produce dangerous hyperkalemia
- Glaucoma, head injury—as it raises intraocular pressure, intracranial pressure, Intra-abdominal pressure, and blood pressure.

**Side effects**

- Malignant hyperthermia – in patients with pseudocholinesterase deficiency
- Hyperkalemia
- Bradycardia
- Muscle fasciculations.

**Q4. What is regional anesthesia? Enumerate its types.****What are the various methods of administering regional anesthesia?**

- Ans.** Regional anesthesia is a procedure which produces loss of sensation in the part for which anesthesia is given without loss of consciousness and without impairment of central control of vital functions.

**Methods**

- Topical (surface or infiltration) anesthesia
  - **Surface anesthesia**
    - Dibucaine, tetracaine, eutectic lignocaine/prilocaine, cocaine, benoxinate, oxethzaine, benzocaine are the commonly used agents.
  - **Infiltration local anesthesia**
    - Used in procedures such as incision and drainage, suturing of lacerated wounds, minor excision procedures, etc. whereby only skin and subcutaneous tissues are anesthetized.
    - Adrenaline is used to prolong action and bicarbonate is used to hasten the onset of action.
    - However, adrenaline cannot be used in regions supplied by end arteries (Nose, Ear, penis, fingers and toes).
- Neuraxial anesthesia (Spinal anesthesia, epidural anesthesia and caudal anesthesia)
- Intravenous regional anesthesia or Bier's block
  - Veins are filled with local anesthetic lignocaine without adrenaline which is kept there by using a tourniquet with pressure above systolic pressure in surgeries of forearm, hand, distal leg and foot. Bupivacaine and etidocaine are contraindicated in this procedure.
- Conduction block (Field block or nerve block)
  - **Field block:** done by giving subcutaneous infiltration to block all the nerve endings in that area.
  - **Nerve block:** Brachial plexus block or wrist block in upper limb, Stellate ganglion block or trigeminal nerve block in head and neck and intercostal nerve block or celiac plexus block in thorax and abdomen.

**Q5. Enumerate the regional/local anesthetic agents and discuss its mechanism of action.****Ans.**

Low potency, short duration	Procaine, chloroprocaine
Intermediate potency and duration	Lignocaine, prilocaine
High potency, long duration	Dibucaine, bupivacaine, ropivacaine, tetracaine
Amide group	Prilocaine, lignocaine, dibucaine, bupivacaine, ropivacaine
Ester group	Procaine, chloroprocaine, benzocaine, tetracaine, cocaine

**Mechanism of action**

- Prolong the inactive state of the cationic form of sodium channel in axon and produce conduction blockade
- Resting nerve is resistant to blockade whereas it develops rapidly when the nerve is repeatedly stimulated
- Small myelinated nerve fibers are blocked earlier
- Bicarbonate increases the onset of action by increasing the unionized form
- Other effects—CNS stimulation followed by depression except in lignocaine which causes drowsiness and lethargy even as early effect. They also cause vasodilatation and

fall in blood pressure (Exception to both is cocaine which causes vasoconstriction and CNS stimulation only.)

#### **Sequence of nerve blockade**

- Pain f/b temperature f/b touch f/b deep pressure
- Bitter f/b sweet f/b sour f/b salty taste.

#### **Benefits of amide anesthetics over esters**

- Produce less hypersensitivity reactions
- Produce rapid, more intense and long lasting anesthesia.

#### **Specific points**

- Most potent, most toxic and longest acting local anesthetic is dibucaine
- Most cardiotoxic and agent with maximum local tissue toxicity is bupivacaine
- Least toxic agent is prilocaine (causes methemoglobinemia) and shortest acting agent is chloroprocaine (contraindicated as spinal anesthetic because it contains neurotoxic agent sodium metabisulphite which can cause paralysis)
- Cocaine is now seldom used and that too only in ocular anesthesia.

### **Q6. Write a note on spinal regional anesthesia.**

#### **Enumerate the complications of spinal anesthesia.**

**Ans.**

- **Site:** L2-3/ L3-4 in adults and L4-5 in children
- **Structures pierced:** Skin, superficial fascia, subcutaneous tissue, supraspinous and intraspinal ligaments, ligamentum flavum, dura and arachnoid. The drug is finally delivered into the subarachnoid space.
- **Level of block:** Motor block is 2 levels lower and autonomic block is 2 levels higher than the sensory block level. Factors affecting the level of spinal anesthesia include the dose of drug, its specific gravity, patient position and site of injection.  
Drugs that are hyperbaric than CSF move towards gravity to settle below CSF and vice versa.
- **Order of block**  
Preganglionic sympathetic f/b temperature f/b pain f/b touch f/b pressure/Deep touch and finally motor sensation.

#### **Contraindications**

##### *Absolute*

- Raised intracranial tension
- Patient denial
- Patients with hemodynamic instability and shock
- Uncontrolled sepsis or sepsis at injection site
- Bleeding disorder or patients on anticoagulant therapy.

##### *Relative*

- Spinal deformity (Kyphosis)
- Uncooperative patient or patient with diseases such as dementia and psychosis which do not allow patient positioning



- Recent myocardial infarction, heart block
- Platelet count < 50,000/mL.

### Drawbacks

- Cannot be used in neck, thoracic, upper abdominal surgeries
- Duration of action cannot be prolonged by any means other than a repeat spinal injection.

### Complications

#### *Intraoperative*

- **Hypotension (m.c.)** – Prevented by preloading with intravenous fluids and managed by putting patient in head low position and rapid intravenous fluid administration and drugs like atropine to counteract bradycardia and vasopressors such as norepinephrine or dopamine to combat severe hypotension not responding to fluid loading.
- **Bradycardia**
- **Cardiac arrest**
- **Respiratory depression**
- **Hypothermia.**

#### *Postoperative*

- **Postlumbar puncture headache (m.c.)**
  - Occurs because of leak of CSF through the puncture site which leads to decreased intracranial pressure
  - Young and pregnant females are more commonly affected
  - Also, large needles with cutting tips and multiple failed attempts increase the risk
  - Onset can be immediate (1–2 hours) or late (48–72 hours)
  - This is usually bilateral frontal or occipital headache that is aggravated by sitting and standing and relieved by lying down
  - Prevention is definitely better than management and prevention is by using a thin pencil point needle
  - Management includes rest in supine position without pillow, IV fluids, caffeine, adequate analgesia
  - Nonresponders can benefit from epidural fluid therapy or epidural venous blood patch (most effective) and lastly vasopressor therapy.
- **Urinary retention**
- **Meningitis, arachnoiditis**
- **Cauda equina syndrome**
- **Cranial nerve palsies** (Abducent nerve most commonly affected. 1,9,10 also affected)
- **Paraplegia.**

**Q7. Write a note on epidural anesthesia.**

**Q8. Write a note on epidural analgesia.**

**Ans.**

- **Site:** Outside the dura using Touhey needle at thoracic/ lumbar/ sacral (caudal) regions.
- **Structures pierced:** Skin, superficial fascia, subcutaneous tissue, supraspinous and interspinous ligaments, ligamentum flavum. The drug is finally delivered into the epidural space.

- It has slower onset of action, is less reliable and is difficult when compared to spinal anesthesia.
- **However, its benefits over spinal anesthesia include,**
  - Duration can be prolonged by additional injection through the epidural catheter
  - Headache is less likely with a true epidural anesthesia
  - Can be used in neck/ thoracic/ upper abdominal surgeries.
- **Uses**
  - Mainly used for control of postoperative pain in patients undergoing prolonged surgeries
  - It is also used in patients with multiple rib fractures to provide analgesia and also to relieve pain associated with malignancies with help of epidural opioids
  - It is also used to provide continuous analgesia by combining local anesthetic with opioids such as fentanyl.
- **Advantages**
  - Less systemic absorption therefore, less toxicity and prolonged duration of action
  - Provide more bloodless field of surgery because of its vasoconstrictive effects.
- **Disadvantages**
  - Can cause elevated blood pressure and arrhythmias due to its vasoconstrictive effects
  - Delay wound healing and can cause local edema, pain and tissue damage due to decreased local blood flow.
- **Contraindications** are same as that of spinal anesthesia.
- **Complications**
  - Hypotension, bradycardia, apnea, unconsciousness and dilated pupils after < 5 minutes of procedure suggest total spinal or high spinal anesthesia which occurs due to inadvertent spinal anesthesia while attempting epidural anesthesia. Management is using intubation and 100% oxygen by mechanical ventilation, IV fluids and vasopressors, atropine and head down position
  - Delayed onset hypotension and apnea suggest subdural extra-arachnoid injection and is managed with IV fluids, vasopressors and oxygen supplementation
  - Meningitis, arachnoiditis and headache are also possible complications but less likely than with spinal anesthesia.
- **Epidural analgesia**

Collection of points from above related to epidural procedure and add the following:

  - Used in patients with rib fractures, chronic cancer pain, prolonged surgeries to reduce postoperative pain and to treat phantom pain as well as many other chronic pain problems
  - Can be used as **patient controlled analgesia** where patient is given a remote control of the epidural catheter with set maximum limit so that patient can self administer the drug as required or **continuous pump analgesia**
  - Opioids are the most commonly used drugs with morphine being the most common agent except in obstetric indications where it can cause respiratory depression
  - Morphine is preferred because it has prolonged duration of action and slow absorption as well as low systemic absorption and therefore no systemic effects
  - Most significant and most common side effect of morphine is respiratory depression and lateral medullary depression. This is not seen with alfentanil and

buprenorphine. Also buprenorphine is more potent than morphine and is now being commonly used.

**Q9. Write a note on brachial plexus block.**

**Ans.**

- Brachial plexus block is a nerve block type of regional anesthesia
- Musculocutaneous nerve, median nerve, radial nerve and ulnar nerve are the main nerves to be blocked when the brachial plexus is considered for block.

**Approaches**

It can be approached through the following approaches,

- **Supraclavicular approach:** Used for procedures of arm, forearm and hand. It provides the most complete brachial blockade from C5-T1. It is quicker to perform. It is given in the area between first rib inferiorly, lateral to lateral border of sternocleidomastoid and just above clavicle and lateral to subclavian artery when done using ultrasound guidance.
- **Infraclavicular approach:** Used for procedures of arm, forearm and hand.
- **Axillary approach:** For procedures from elbow to hand in C7-T1 distribution. It is the safest of the 4 approaches. The injection is given just anterior to the axillary pulsation. Triple injection technique for blocking musculocutaneous, radial and median nerves is the best technique.
- **Interscalene approach:** For procedures of clavicle, shoulder, arm and forearm in C5-7 distribution. The block is given at the level of cricoid cartilage between anterior and middle scalene muscles.

The needle is passed lateral to subclavian artery.

**Indications**

It can be used in all the procedures which meet the following criteria:

- Surgery from shoulder to fingers
- The operation does not require intraoperative nerve studies
- No infection at the intended injection site, no significant bleeding disorder, and no hypersensitivity to local anesthetics.

**Complications**

- **Interscalene approach:** Temporary diaphragmatic paralysis.
- **Supraclavicular approach:** Pneumothorax. Diaphragmatic paralysis is less common complication.
- Intra-arterial or intravenous injection can lead to systemic toxicity – seizures, CNS depression, coma, bradycardia or cardiac arrest.

**Contraindications**

- **Interscalene approach:** Opposite phrenic nerve paralysis/chronic obstructive pulmonary disease.

**Q10. Write a note on caudal block.**

**Ans. Indications for a caudal block**

- Skin grafting
- Hernia repair
- Procedures on the anus and rectum

- Lower limb surgery
- Genitourinary procedures
- Obstetric analgesia for instrumental deliveries.

**Caudal blocks are not common in adults** because lumbar and thoracic epidural space is easier to access in the adult and sacral hiatus and caudal space difficult to access.

- The caudal epidural space is the lowest portion of the epidural system and is entered through the sacral hiatus. The contents of the sacral canal include dural sac between S1-3, filum terminale, sacral and coccygeal nerves, sacral epidural venous plexus and epidural pad of fat
- The distance from the tip of the coccyx to the sacral hiatus is approximately the same as the distance from the tip of the index finger to the proximal interphalangeal joint
- Drugs – Lidocaine 1% and bupivacaine 0.25%. Fentanyl and morphine can be used in caudal blocks also.

### Complications

- Dural puncture and total spinal anesthesia or high spinal anesthesia (discussed in SN on epidural anesthesia)
- Intravascular or intrathecal Injection—seizures, respiratory arrest and/or cardiac arrest.
- Sepsis
- Rectal perforation
- Urinary retention
- Hematoma.

## Q11. Write a note on cervical blocks.

### Ans. Indications

- Thyroidectomy
- Lymph node biopsy or excision
- Carotid endarterectomy
- Plastic surgical procedures
- Procedures on neck/ Supraclavicular region

**Drugs used:** Lignocaine or Bupivacaine

### Nerves blocked

Cutaneous branches to superficial posterior occiput, neck, shoulder, clavicle, jaw, branches to ansa cervicalis and to prevertebral neck muscles so as to block lateral occiput, anterior/posterior neck and shoulder and supraclavicular region.

### Technique

- **Deep Cervical Approach**
  - Produces both motor and sensory blockade.
- **Superficial Cervical Approach**
  - Produces sensory blockade with less complications compared to deep approach.

### Complications

- Dural puncture and subarachnoid injection and total spinal anesthesia or high spinal anesthesia (discussed in SN on epidural anesthesia)

- Injury to vertebral artery, carotid artery, internal jugular or external jugular vessels
- Phrenic nerve, recurrent laryngeal nerve or vagal nerve block
- Brachial nerve plexus blockade
- Hematoma
- Should be avoided in pregnancy as inadvertent absorption has been seen to result in adverse effects on fetus.

**Q12. What is nerve block? What is field block? Discuss the uses of field block anesthesia.**

**Ans. Nerve block** is as the term suggests, block of transmission along a nerve using a local anesthetic or steroid injection. Always remember that the injection is along the nerve and not into the nerve.

**Neurolysis or neurolytic block** is chemical nerve damage using alcohol, cryoprocures or thermal ablation to produce prolonged or permanent block.

**Neurectomy** is cutting a nerve to produce permanent block.

**Field block**

- Done by giving subcutaneous infiltration to block all the nerve endings in that area by giving injections in the border of the surgical area. The actual surgical field is not injected with anesthesia and this differentiates it from infiltration anesthesia.
- It is frequently used at end of surgical procedure to manage postoperative pain for 6–8 hours
- Multiple injections of local anesthetic are required in the border or the surgical area to produce field block
- Anesthesia from a field block lasts longer than that from local infiltration, and it does not cause swelling in the surgical field or obscure local anatomy.
- **Uses**
  - Ingrowing toenail excision
  - Abscess incision and drainage
  - Removal of foreign body
  - Incision and drainage of paronychia
  - Before endoscopic procedures
  - Subcutaneous lesion excision
- **Complications**
  - Drug allergy
  - Injection site infection, abscess, hematoma
  - Inadvertent systemic injection and cardiovascular and central nervous system toxicity.

**Q13. Enumerate the drugs used as general anesthetics and discuss the properties and uses of halothane.**

**Write a note on propofol.**

**Ans. General anesthetics commonly used are as follows:**

Inhalational		Intravenous	
Gas	Liquid	Induction agents	Slow acting agents
<b>Nitrous oxide</b> (colorless, odorless, nonirritating. Produce second gas effect on induction and diffusion effect on weaning. Has hematologic/ neurologic toxic effects on long term administration due to interaction with vitamin B <sub>12</sub> )	<b>Ether</b> (maximum muscle relaxant property Contraindicated in diabetics Volatile, explosive)	<b>Thiopentone</b> (very short acting, IV agent of choice in neurosurgical procedures** Is poor analgesic and poor muscle relaxant Extravasation produce hyperalgesia, necrosis and gangrene Contraindicated in patients with porphyria, cardiac and respiratory diseases)	Benzodiazepines
	<b>Halothane</b> (Inhalation Agent of choice in asthma patients)**	Propofol	Opioids
	<b>Isoflurane</b> (Inhalation Agent of choice in cardiac patients, myasthenia and day care surgery)** <b>Desflurane</b> (Both are nonarrhythmogenic, do not provoke seizures and have no liver/ kidney toxicity)	<b>Etomidate</b> (IV Agent of choice in cardiovascular surgeries)**	Ketamine
	<b>Sevoflurane</b> (Drug of choice in children)	<b>Ketamine</b> (Dissociative anesthesia IV Agent of choice in children, postpartum hemorrhage, asthma and in emergency situation.** Produce emergence reaction in schizophrenics)	
	<b>Methoxyflurane</b> (cause high output renal failure, not used)		

### Halothane

- It is volatile, with sweet odor, nonirritating and noninflammable
- It is the most potent inhalational agent with poor analgesia and poor muscle relaxation properties
- It is suitable for induction and maintenance in children and adults.

### Effects

#### Cardiovascular system

- Direct depression of myocardial contractility by decreasing intracellular calcium
- Decrease in heart rate due to vagal stimulation
- Sensitize heart to arrhythmogenic action of adrenaline.

*Respiratory system*

- Ventilation – perfusion mismatch
- Bronchodilatation (agent of choice in asthmatics)
- Respiratory depression.

**Hepatotoxic and can cause malignant hyperthermia**

**Inhibits intestinal and uterine contractions** and is therefore the agent of choice during internal/external versions in late pregnancy.

**Problems**

- Contraindicated in labor
- Produce postanesthetic chills and shivering.

**Propofol**

- Most frequently used intravenous anesthetic today
- Used for both induction and maintenance.

**Advantages**

- Antiemetic effect
- Smooth and rapid induction
- Relatively short duration of action
- Antipruritic action.

**Disadvantages**

- Causes a fall in blood pressure and bradycardia
- No anticonvulsive action
- Increase in incidence of life threatening infections due to decreased neutrophil chemotaxis but no effect on phagocytosis
- No muscle relaxation action.

**Uses**

- Day care surgery
- Total intravenous anesthesia regime (TIVA) supplemented by fentanyl
- Agent of choice or anesthesia in ICU and in patients with malignant hyperthermia.

**Drawback**

- Cannot be used in children and in adults for duration > 48 hours especially in critical care units as it can lead to **propofol infusion syndrome** which occurs due to failure of free fatty acid metabolism and mitochondrial respiratory chain. It can manifest as metabolic acidoses, cardiomyopathy, hepatomegaly, or multi-organ failure.

**Q14. Write a note on the role and indications of postoperative ventilation.**

**Ans.**

**Benefits of postoperative ventilation are as follows:**

- Airway protection and prevention of aspiration
- Improved ventilation and good oxygenation
- Decreased work of breathing

- Avoidance of hypercarbia and hemodynamic instability in sedated patients
- Direct endotracheal access for removal of secretions.

**Consideration of elective postoperative ventilation is done in patients under following circumstances**

- Patients not able to maintain a patent airway because of residual anesthetic effect, cerebral ischemia/injury, hyponatremia, congestion after reconstructive surgery, vocal cord paralysis or anaphylaxis
- Patients with limited muscle excursion due to upper abdominal/ thoracic surgery, residual neuromuscular relaxation, hypoglycemia, hyponatremia and acidoses
- Patients with respiratory compromise due to excessive atelectasis, acute respiratory distress syndrome, or chronic obstructive pulmonary disease
- Intraoperative uncontrolled hypertension, myocardial ischemia/infarction, or arrhythmias, hypoadrenalism are also indicators for continued postoperative ventilation till the condition stabilizes prior to extubation
- Patients with pulmonary edema, pulmonary embolism, pulmonary hypertension or kyphoscolioses
- Any patient with rising  $\text{EtCO}_2$  or Tobin index  $> 105$  breaths/min/L (rapid shallow breathing index or tobin index = Respiratory rate/tidal volume)
- Patients with coagulopathy or hypothermia during surgery should have these corrected prior to extubation
- Hypoglycemia, hyponatremia, hypokalemia, hypocalcemia
- Patients with raised intracranial pressure or probability of cerebral edema or raised intracranial pressure in postoperative period
- Prevent the disturbance of a large skin area after grafting, sometimes elective sedation and ventilation is advised for first 48 hours
- Some patients of prolonged cardiac surgeries, esophageal surgeries and neurosurgeries are also considered for elective intubation
- In summary, any patient who does not meet the extubation criteria at the end of surgery and any patient who is anticipated to have one of the above feature should be considered for elective intubation
- Points not favoring extubation include:
  - Vital capacity  $< 10$  mL/kg
  - Minute ventilation  $> 10$  mL
  - Inspiratory pressure  $> 25$  cm  $\text{H}_2\text{O}$
  - Rapid shallow breathing index/Tobin index (Respiratory rate/Tidal volume)  $> 105$
  - Tidal volume  $< 5$  mL/kg.

**Q15. Enumerate the complications of general anesthesia.**

**Ans.**

**Complications of general anesthesia are as follows:**

- Respiratory problems
  - Trauma to airway, lips, tongue
  - Laryngeal edema



- Tooth damage, mandibular dislocation
- Sore throat
- Atelectasis and chest infection
- Cardiovascular problems
  - Hypercarbia, hypoxia
  - Hypo/hypervolemia
  - Myocardial ischemia/infarction
  - Bradycardia
  - Cardiac arrest
- Neurological complications
  - Cerebral hypoperfusion
  - Cerebral depression due to anesthetic drugs
  - Postoperative delirium, confusion or amnesia
  - Anesthesia awareness
- Postoperative nausea and vomiting (PONV), headache, shivering, dizziness
- Drug allergy/ anaphylaxis
- Acid-base and electrolyte imbalances.

**Q16. Write a note on central venous pressure monitoring and its role in surgery.**

**Ans.**

- **Central venous pressure or right atrial pressure** is the measurement of pressure at the junction of superior vena cava and right atrium and gives a measure of blood volume status in body and venous capacitance status as well as the measure of **right ventricular preload**.
- **Normal value**—5–12 cm H<sub>2</sub>O and it is 5 cm H<sub>2</sub>O higher in patients on ventilator.
- Subclavian vein or internal jugular vein are used for cannulation for central line and CVP measurement (Subclavian route is preferred).
- **Measurement**
  - Clinical method is by measuring the jugular venous pulse height in a patient positioned in semireclined position.
  - The other bedside method is by using CVP manometer or automated electronic pressure monitor connected to a central line.
  - CVP should be measured at end expiration to avoid respiratory variation on CVP value.
- **Waveform**
  - It has 3 positive (a, c, v) and 3 negative (x, y, z) waves.
  - **The A wave** starts just after the P wave of ECG ends and represents the atrial contraction or “atrial kick”. The high point of the A wave is the atrial pressure at maximum contraction and is a good point to measure CVP.
  - **The c-wave** occurs at closure of the tricuspid valve and represents the ventricular contraction thus, always occurring after the R wave of ECG. The crest of the c-wave is the atrial pressure increase caused by the tricuspid valve bulging back into the atrium.
  - **The X wave** is the descent in atrial pressure that occurs during ventricular contraction. The descent on either side of c wave are called x and x’ respectively.

- **The v wave** indicates atrial filling in late systole with closed tricuspid valve and peaks after the peak of T wave.
- The decline in atrial pressure on opening of tricuspid valve is **y wave**.
- **The Z-point** occurs just before closure of the tricuspid valve, coincides with the middle to end of the QRS wave and is a good indicator of right ventricular end diastolic pressure. It is useful when A waves are not visible, as in atrial fibrillation.
- Waveform abnormalities can result from arrhythmias, tricuspid valve diseases (Stenosis or regurgitation) and atrial septal defects as can be made out from the discussion above.
- **Factors affecting CVP**
  - Blood volume in circulation
  - Venous return
  - Cardiac performance status
- **Causes of decreased CVP**
  - Hypovolemia
  - Distributive shock
- **Causes of elevated CVP**
  - Volume overload
  - Tension pneumothorax
  - Cardiac tamponade
  - Pulmonary hypertension
  - Positive pulmonary pressure ventilation
  - Congestive cardiac failure
  - Pulmonary embolism
  - Pleural effusion
  - Change from standing to supine position
- **Uses**
  - To guide fluid therapy
  - Guide use of vasopressors and colloid infusions
  - Diagnoses of arrhythmias. For example, in atrial fibrillation, the 'a' wave of CVP disappears and 'c' wave becomes taller
  - Diagnoses of tricuspid valve lesions (regurgitation or stenosis), pulmonary hypertension, pulmonary valve stenosis
  - The catheter is also useful for rapid fluid infusion, infusion of hypertonic solutions and medications that could damage the peripheral vessels as well as for collection of blood for investigations.

## SKIN

**Q17. Write a note on premalignant skin lesions.**

**Ans. Squamous cell cancer**

- **Actinic keratosis/Arsenic keratosis:** 20% risk of malignancy
- **Bowen disease:** Erythematous scaly plaque or patch especially in patients with chronic solar damage or arsenic exposure.

- Cutaneous horn (when the lesion has height > width) – 10% risk of malignancy.
- **Discoid lupus erythematosus, dystrophic epidermolysis bullosa**
- **Erythema ab igne**
- **Erythroplakia of Queyrat:** Bowen's disease of penis. Red velvety patch or plaque on penis.
- Epidermodysplasia verruciformis
- Human papilloma virus infected genital warts
- **Keratoacanthoma:** Symmetrical cutaneous growth with a central crater filled with keratin plug. It has 2:1 male preponderance with increased risk in patients infected with human papilloma virus, smoking and chemical carcinogens. It grows to 1–3 cm in few weeks time and then resolves. The removal of central keratin plug produces healing.

#### Basal cell cancer

- **Nevus sebaceous of Jadassohn:** Looks like linear verrucous nevi and has 10% risk of basal cell cancer.

#### Malignant melanoma

- Lentigo maligna (Hutchinson melanotic freckle)
- Giant congenital pigmented nevi
- Dysplastic nevi

#### Adenocarcinoma

- Extramammary Paget's Disease – Presents as eczematous lesion/ intertrigo in genitalia, Perianal, axillary regions. It is associated with intraepidermal adenocarcinoma in 25% cases.

### Q18. Write a note on squamous cell cancer. Discuss its management in brief.

**Ans.** It is the second most common malignancy after basal cell cancer.

Also known as

- **Kangri cancer** SCC of abdomen or back of thighs due to charcoal burner in Kashmir.
- **Kang cancer** in Tibet due to sleeping on oven bed.
- **Chimney sweeper cancer** scrotal skin in chimney sweepers.
- **Marjolin ulcer**
  - SCC at sites of chronic ulcers (venous ulcers, radiation injury, thermal injury)/ sinus tracts (osteomyelitis)/keloids/scars
  - So, it is always necessary to take a wedge biopsy or excision biopsy to rule out Marjolin in a patient with chronic nonhealing ulcer
  - This form of SCC has no nerves, no lymphatics, no blood supply, no skin appendages
  - Therefore, it is painless, is nonmetastatic and is radioresistant
  - Therefore, the treatment is plain surgical excision or amputation
  - Rest features remain the same.

#### Risk factors

- Ultraviolet radiation (UV B > UV A)

- Arsenic exposure
- Ionizing radiation
- Human papilloma virus infection
- Tobacco
- Chronic ulcers/sinus tracts/keloids/scars—Marjolin ulcer
- Polycyclic aromatic hydrocarbons
- Genetic – Albinism, Bazex syndrome, epidermolysis bullosa, xeroderma pigmentosum.

### Precursor lesions

- **Actinic keratosis/Arsenic keratosis:** 20% risk of malignancy
- **Bowen disease:** Erythematous scaly plaque or patch especially in patients with chronic solar damage or arsenic exposure
- Cutaneous horn (when the lesion has height > width): 10% risk of malignancy
- **Discoid lupus erythematosus, dystrophic epidermolysis bullosa**
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- **Erythroplakia of Queyrat:** Bowen's disease of penis. Red velvety patch or plaque on penis
- Epidermodysplasia verruciformis
- Human papilloma virus infected genital warts
- **Keratoacanthoma:** Symmetrical cutaneous growth with a central crater filled with keratin plug. It has 2:1 male preponderance with increased risk in patients infected with human papilloma virus, smoking and chemical carcinogens. It grows to 1–3 cm in few weeks time and then resolves. The removal of central keratin plug produces healing.

### Clinical feature of SCC lesion

- Ulcer with everted or rolled out edge
- Floor is covered by gray-white slough
- Base is indurated and may be fixed to underlying structures.
- Spread is by local extension or lymphatics.

### Histopathological types

- Cauliflower type (Proliferative)
- Ulcerative
- Verrucous (Ackerman tumor)
- Plaque like.

**Keratin cell nests (Epithelial pearls)** are the histopathological hallmark of SCC. Exceptions where these are not seen include bladder SCC, esophagus SCC and rapidly growing SCC.

### Imaging features

- CT with IV contrast is the investigation of choice
- MRI is used when there is doubt of intracranial/orbital/parapharyngeal extension

- PET is used for evaluation of suspicious distant metastasis and is more important for evaluation of merkel cell cancer than in SCC
- Excision biopsy for smaller lesions and wedge biopsy for large lesions.

### TNM Staging

T1	< or = 2 cm
T2	> 2 cm
T3	Muscle, cartilage, nonaxial bones or jaw involvement
T4	Axial skeleton involvement, skull base involvement
N1	< or = 3 cm, single, ipsilateral node
N2a	> 3 to < or = 6 cm, single, ipsilateral node
N2b	> 3 to < or = 6 cm, multiple, ipsilateral node
N2c	> 3 to < or = 6 cm, contralateral, bilateral
N3	> or = 6 cm node
M0	No metastasis
M1	Metastatic disease

### Staging

I	T1N0
II	T2N0
III	Any T, N1 T3N0
IV	T4 any N Any T, N2/3 Any T, any N, M1

### Management

<b>Management of primary</b>	<p><b>In situ lesions</b></p> <ul style="list-style-type: none"> <li>• Cryotherapy</li> <li>• Narrow field radiotherapy</li> <li>• Topical 5-Fluorouracil or Imiquimod 5%.</li> </ul> <p><b>Stage I/II</b></p> <ul style="list-style-type: none"> <li>• Surgery is the definitive management.</li> <li>• Margin is 4 mm for tumor up to 2 cm and 6 mm for tumor more than 2 cm size</li> <li>• Moh's micrographic surgery is used in difficult areas with comprehensive frozen section analysis</li> </ul> <p><b>Stage III</b></p> <ul style="list-style-type: none"> <li>• En-bloc resection of tumor with fat/ muscle/ bone/ orbit</li> <li>• Primary radiotherapy with concomitant radiotherapy when surgery is not possible.</li> </ul>
<b>Management of nodes</b>	<p><b>N+ disease</b></p> <ul style="list-style-type: none"> <li>• Therapeutic nodal dissection</li> <li>• Modified radical neck dissection is now preferred over radical neck dissection in case of neck nodes.</li> <li>• Monoblock dissection is preferred.</li> <li>• Sentinel lymph node biopsy is now being evaluated in SCC cases also.</li> </ul>

Contd...

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<b>Adjuvant treatment</b>	<b>Radiotherapy indications</b> <b>T</b> <ul style="list-style-type: none"> <li>• Perineural invasion</li> <li>• Close or positive margins</li> <li>• Tumor satellites</li> </ul> <b>N</b> <ul style="list-style-type: none"> <li>• Node &gt; 3 cm size</li> <li>• 2 or more nodes involved</li> <li>• Parotid node positive</li> <li>• Extranodal invasion</li> </ul> <b>Concurrent chemotherapy</b> with Carboplatin/5-Fluorouracil/ Cisplatin is preferred over sequential regimes. But the role is still investigational. No neoadjuvant role.
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**Q19. Write a note on basal cell carcinoma.**

**Ans.** It is the most common skin malignancy.

Arise from the basal cells of the epidermis of the skin.

Also known as

- **Field fire cancer:** because of its habit of subsiding at one side erupting at other site.
- **Rodent ulcer:** because of its habit of penetrating deeper tissues like a rat.
- **Tear cancer:** because of its predilection for the areas which are normally covered by tears when a person cries i.e. medial canthus, nasolacrimal folds, medial cheeks.

**Risk factors**

- Ultraviolet radiation (UV B > UV A)
- Arsenic exposure
- Ionising radiation
- Genetic—Gorlin syndrome, Bazex syndrome, epidermolysis verruciformis, xeroderma pigmentosum, nevoid basal cell carcinoma syndrome.

**Precursor lesions**

- **Nevus sebaceous of Jadassohn:** Looks like linear verrucous nevi and has 10% risk of basal cell cancer

**Clinical feature of BCC lesion**

- Appears as a nodule or papule from the surface of which the epidermis peels off and results in an ulcer with raised, rolled out, beaded edge
- Floor can reach upto bone
- Base is indurated and may be fixed to deeper structures
- Spread is by local extension or lymphatics.

**Histological subtypes**

- Noduloulcerative type is the most common
- Basosquamous
- Pigmented
- Superficial
- Morpheic

**Imaging features**

- CT with IV contrast is the investigation of choice
- MRI is used when there is doubt of intracranial/ orbital/parapharyngeal extension
- PET is used for evaluation of suspicious distant metastasis
- Excision biopsy for smaller lesions and wedge biopsy for large lesions.

**TNM Staging**

T1	< or = 2 cm
T2	> 2 cm
T3	Muscle, cartilage, nonaxial bones or jaw involvement
T4	Axial skeleton involvement, skull base involvement
N1	< or = 3 cm, single, ipsilateral node
N2a	> 3 to < or = 6 cm, single, ipsilateral node
N2b	> 3 to < or = 6 cm, multiple, ipsilateral node
N2c	> 3 to < or = 6 cm, contralateral, bilateral
N3	> or = 6 cm node
M0	No metastasis
M1	Metastatic disease

**Staging**

I	T1N0
II	T2N0
III	Any T, N1 T3N0
IV	T4 any N Any T, N2/3 Any T, any N, M1

**Management**

<b>Management of primary</b>	<b>In situ lesions</b> <ul style="list-style-type: none"> <li>• Cryotherapy</li> <li>• Narrow field radiotherapy</li> <li>• Topical 5-Fluorouracil or Imiquimod 5%</li> </ul> <b>Stage I/II</b> <ul style="list-style-type: none"> <li>• Surgery is the definitive management</li> <li>• Margin is 3 mm</li> <li>• Moh's micrographic surgery is used in difficult areas with comprehensive frozen section analysis</li> </ul> <b>Stage III</b> <ul style="list-style-type: none"> <li>• En-bloc resection of tumor with fat/ muscle/ bone/ orbit</li> <li>• Primary radiotherapy with concomitant radiotherapy when surgery is not possible</li> </ul>
<b>Management of nodes</b>	<b>N+ disease (Clinical or radiological)</b> <ul style="list-style-type: none"> <li>• Therapeutic nodal dissection</li> <li>• Modified radical neck dissection is now preferred over radical neck dissection in case of neck nodes</li> <li>• Monoblock dissection is preferred</li> <li>• Sentinel lymph node biopsy is now being evaluated in SCC cases also</li> </ul>

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<b>Adjuvant treatment</b>	<b>Radiotherapy indications</b> <b>T</b> <ul style="list-style-type: none"> <li>• Perineural invasion</li> <li>• Close or positive margins</li> <li>• Tumor satellites</li> </ul> <b>N</b> <ul style="list-style-type: none"> <li>• Node &gt; 3 cm size</li> <li>• 2 or more nodes involved</li> <li>• Parotid node positive</li> <li>• Extranodal invasion</li> </ul> <b>Concurrent Chemotherapy</b> with Carboplatin/5-Fluorouracil/Cisplatin is preferred over sequential regimes. But the role is still investigational. No neoadjuvant role
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**Q20. Write a note on pigmented nevus.****Q21. Write a note on melanocytic nevi and discuss its malignant potential.****Ans.** Pigmented nevus are of following types

<b>Congenital</b> (Risk of malignancy in non-giant varieties is 1–5%)	Small	<1.5 cm
	Intermediate	1.5–10 cm
	Large	>10 cm
	Giant	> 20 cm Risk of malignancy 5–10% Risk of leptomeningeal melanosis and nonreducible risk of CNS melanoma, seizures and hydrocephalus Therefore these patients should always have MRI Brain Most common site is trunk. ( <b>Hairy nevi/Bathing trunk nevi</b> )
<b>Acquired</b>	Junctional	Seen on soles, palm, digits and genitalia Occur before puberty and have definite malignant potential
	Intradermal (Blue nevus, Mongolian spot, common mole)	Seen on buttocks, face, dorsum of foot and are not premalignant
	Compound (Spitz nevus or juvenile melanoma)	Premalignant but less than junctional nevus
	Dysplastic nevus	Can be sporadic or inherited in an autosomal dominant pattern with incomplete penetrance They have a high risk of malignancy
	Lentigo maligna (Hutchinson melanotic freckle)	Seen on face, neck, conjunctive and oral cavity. Are premalignant
	Ota nevus	More common in women Occur along trigeminal nerve V1, V2 distribution
	Ito nevus	Occur in shoulder region. Usually seen with Ota nevus



**Q22. Write a note on malignant melanoma.**

**Ans.** Arise from the basal cells of the epidermis of the skin called the melanocytes.

**Risk factors**

- Strong family history
- Past history of melanoma
- Immunosuppression
- Genetic—familial atypical multiple mole melanoma syndrome (CDKN2A mutation on Ch.9), albinism, xeroderma pigmentosum
- Fair skin individuals with history of prolonged sun exposure.

**Precursor lesions**

- Lentigo maligna (Hutchinson melanotic freckle)
- Giant congenital pigmented nevi
- Dysplastic nevi.

**Clinical feature of melanoma lesion**

- More common in females in third decade of life
- Can occur in cutaneous, mucosal (anorectal, genital) and ocular (choroid, iris, ciliary) forms. Can also occur in leptomeninges, nasopharynx and paranasal sinuses
- Can be black, brown, blue or grey or any mix of colors
- The lesions have irregular borders, any variety of surface, is nontender and is fixed with skin but free from deeper structures
- Spread is by local extension and satellite lesions, lymphatic or blood routes.

**Histological subtypes**

- Superficial spreading is the most common variety
- Nodular
- Acral lentiginous
- Desmoplastic
- Amelanotic melanoma
- Other.

**Features suggestive of malignancy in a pre-existing mole**

- Asymmetry/nodularity
- Border irregularity
- Color variation
- Diameter > 6 mm/Rapid increase in size
- Elevated surface
- Swellings (lymphadenopathy)
- Surface (Scaling, erosion, oozing, crusting, loss of hair, nodularity)
- Surrounding (Satellite lesions, pigmented halos, nodules)
- Symptoms (Awareness of lesion change, pain, itching, discharge, bleeding)
- Microscopy (Mitoses, Pleomorphism, Subepithelial invasion, hyperchromatism, anaplasia)

**Features of various types of melanoma**

<b>Superficial spreading</b>	<ul style="list-style-type: none"> <li>• m.c. type.</li> <li>• Arise from pre-existing dysplastic nevus more commonly on trunk in males and lower leg and back in females</li> <li>• Lateral growth predominates</li> </ul>
<b>Acral lentiginous</b>	<ul style="list-style-type: none"> <li>• Most common type in dark skinned individuals</li> <li>• Most aggressive and least common in white skinned individuals</li> <li>• Arise from palm, sole, mucosal surfaces, subungual location mainly from great toe/ thumb nails. Hutchinson sign is seen in the subungual variety</li> </ul>
<b>Lentigo maligna melanoma</b>	<ul style="list-style-type: none"> <li>• It has lowest metastatic potential but has poor prognosis. Its in-situ form is called Hutchinson freckle</li> <li>• Common on face in women</li> </ul>
<b>Nodular melanoma</b>	<ul style="list-style-type: none"> <li>• It is second most common after SS melanoma</li> <li>• It has worst prognosis</li> <li>• Arise from head, neck or trunk</li> <li>• Vertical growth predominates</li> </ul>

**Investigations**

- Biopsy of lesion and any palpable and suspicious lymph nodes or satellite nodules – incisional or excision to establish the diagnoses
- Chest X-ray
- Liver function test
- Markers such as Melan A, S-100, HMB 45 and LDH
- PET is used for evaluation of suspicious distant metastasis.
- **Sentinel lymph node biopsy** is performed for all melanomas >1 mm thick and all melanomas of any thickness with clark level IV or V and high mitotic count.

**Clark staging**

- Upto epidermis only
- Into papillary dermis
- At the interface of papillary dermis and reticular dermis
- Into the reticular dermis
- Into the subcutaneous tissue.

**Breslow staging**

- < or = 0.75 mm
- > 0.75 mm to < or = 1.5 mm
- > 1.5 mm to < or = 2.25 mm
- > 2.25 mm to < or = 3 mm
- > 3 mm.

**Management**

<b>Management of primary</b>	<p>Surgery is the definitive management.</p> <p><b>Margins</b></p> <ul style="list-style-type: none"> <li>• <b>In situ lesions or lentigo maligna</b> – 0.5 cm</li> <li>• <b>Lesion &lt; or = 1 mm</b> – 1 cm</li> <li>• <b>Lesion 1–4 mm</b> – 2 cm</li> <li>• <b>Lesion &gt; or = 4 mm or satellitoses</b> – 3–5 cm with high dose interferon alfa therapy postoperatively</li> </ul> <p><b>Moh's micrographic surgery</b> is used in difficult areas with comprehensive frozen section analysis</p>
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<b>Management of nodes</b>	<b>N+ disease</b> Therapeutic monoblock nodal dissection <b>N0 disease</b> Sentinel lymph node biopsy as mentioned above.
<b>Adjuvant treatment</b>	Melanoma is chemoresistant and radioresistant. However single agent or multiple agent chemotherapy is still used in metastatic melanoma. Agents include Dacarbazine, Vincristine and Melphalan. Also isolated limb chemoperfusion using Melphalan is tried in recurrences and advanced stage disease. The only adjuvant therapy of proven benefit is <b>high dose interferon alfa</b> <b>Indications</b> Melanoma > or = 4 mm Melanoma with satellitoses Lymph node positive cases <b>Dose</b> Monthly injections <b>IV</b> in 1st month followed by subcutaneous for next 11 months Has shown improved disease free survival and overall survival

## BURNS AND PLASTIC SURGERY

**Q23. Write a note on skin grafts and flaps.**

**Q24. Differentiate between skin grafts and flaps.**

**Differentiate between full thickness and partial thickness skin grafts.**

**Q25. Enumerate the phases of 'take' of graft.**

**Q26. Classify flaps used in plastic reconstructive surgery.**

**Ans.**

### **Reconstructive surgery ladder**

- The options of wound closure have evolved with mankind. The wound healing by secondary intention was initially used which was progressively changed to wound healing by primary intention, skin grafting, local and distant tissue transfer (Flaps), and finally free tissue transfer and now skin regeneration and stem cell techniques. These constitute the **Reconstructive ladder**.

### **Blood supply to the skin**

- **Deep vessels** arise from the aorta and is the main arterial supply to the **head, neck, trunk and limbs**
- **Interconnecting vessels include fasciocutaneous (or septocutaneous)** perforating vessels which supply to the skin in the **limbs** and **musculocutaneous vessels** which supply to the skin of the **torso**
- **Plexuses in skin** include subepidermal and dermal plexus (thermoregulatory), subdermal plexus, subcutaneous plexus (fasciocutaneous) plexus or prefascial and subfascial (musculocutaneous) plexus.

**Classification of skin grafts**

Skin grafts are detached portion of skin without its own blood supply that is attached at some other site where it attaches at the recipient site and forms new blood vessels with help of recipient site.

**Types of skin grafts include the following**

*Based on thickness*

- **Split thickness skin graft:** Includes epidermis and part of dermis only
  - Thin: Thiersch graft (0.006–0.012 inches)
  - Intermediate: Blair graft (0.012–0.018 inches)
  - Thick: Padgett graft (0.018–0.024 inches)
- **Full thickness skin graft:** Includes epidermis and entire dermis. It is called Wolfe graft
- **Composite graft:** Contains subcutaneous tissue/cartilage/muscle/bone with skin.

*Based on donor*

- **Autograft:** from some site in the recipient itself
- **Allograft:** from other person of same species
- **Xenograft:** from other person of some other species.

**Classification of flaps**

Flaps contain its own blood supply with itself and does not depend on the recipient site for the blood supply.

*Based on skin Circulation*

- **Random flaps**
  - No directional blood supply
  - Not based on any known vessel.
- **Axial flaps**
  - Named artery running along the axis of the flap.
  - Example: The deltopectoral flap based on perforating vessels of the internal mammary artery.
- **Fasciocutaneous Axial flaps**
  - Based on vessels running either within or near the fascia and is mainly used in limbs.
- **Musculocutaneous Axial flaps**
  - Based on perforators that reach the skin through the muscle and is mainly used in torso.
- **Venous Axial flaps**
  - Based on venous rather than arterial pedicles
  - Example: saphenous flap based on the short saphenous vein and often used to reconstruct defects around the knee.

*Based on Methods of Transferring Flaps*

- **Advancement**

Stretching of the flap: Excision of Burow's triangles at its base

  - V-Y or rectangular advancement

- Z-plasty at its base
- A combination of the above.
- **Transposition**
  - The flap is moved into a defect from an adjacent position, leaving a defect which must be closed by another method.
- **Rotation**
  - The flap is rotated into the defect.
- **Rhomboid or Limberg flap**

*Based on Composition*

- Cutaneous
- Fasciocutaneous
- Fascial
- Musculocutaneous
- Muscle only
- Osseocutaneous
- Osseous
- Omental, jejuna
- Perforator flap.

*Based on Contiguity*

1. **Local, regional and distant flaps**

- **Local flaps**
  - These are composed of tissue adjacent to the defect.
- **Regional flaps**
  - These are composed of tissue from the same region of the body as the defect.
- **Distant flaps**
  - Pedicled distant flaps are from a distant part of the body to which they remain attached.

2. **Pedicled flap or free flap**

- **Free flaps**
  - These are completely detached from the body and anastomosed to recipient vessels close to the defect.

**Difference between skin grafts and flaps is as follows:**

Skin grafts	Flaps
Depend on the recipient site for blood supply	Carry their own blood supply with them
It requires a good vascular recipient bed to survive	Can survive at recipient sites with low blood supply
Can be partial thickness or full thickness or composite grafts	Can be random or axial or free flaps
Easy to construct and takes less time	Difficult and skilful procedure requiring more time and planning
Irradiated wounds are not well served	Irradiated wounds can be covered nicely

*Contd...*

Skin grafts	Flaps
Open wounds with exposed bone, cartilage or tendon especially when without periosteum, perichondrium or peritenon respectively are a relative contraindication	All these sites can be taken care of by flaps.
Xenografts and allografts are possible	Not possible

#### Difference between partial thickness and full thickness grafts:

Full thickness skin grafts	Partial thickness skin grafts
Contain entire dermis and epidermis	Contain epidermis and part of dermis
Preferred for nasal tip, ear concha or rim of helix, medial canthus of eye, and eyelids.	Burnt areas, Chronic superficial ulcers and temporary covering are indications
Cover less surface area and seldom survive in precarious conditions	Cover larger surface area and survive in precarious conditions
Slow in healing and take	Heal rapidly and takes quickly
Cosmetic superiority	Cosmetically inferior
More resistant to trauma	Least resistant to trauma
Maximum primary contraction Less secondary contraction	Less primary contraction Maximum secondary contraction
Good sensation	Poor sensation
Resembles native skin	Less resemblance to native skin

(**Primary contraction** is directly related to dermis content and **Secondary contraction** is due to underlying muscles. Because of more dermis, full thickness graft have more primary contraction and vice versa)

#### Process of 'take' of graft

- **Phase of imbibition**
  - First 36–48 hours
  - Nutrition is obtained through diffusion from recipient bed.
- **Phase of inosculation**
  - Up to 3 days
  - Capillaries in graft orient with recipient vessels and start anastomosing.
- **Phase of revascularization**
  - 3–5 days
  - Graft vascularized by new vessels growing into it from the recipient bed.

#### Factors adversely affecting graft survival

##### Local

- Infected recipient site
- Postoperative infection
- Seroma, hematoma
- Necrotic material or foreign body
- Lack of immobilization of the graft area.

*Systemic*

- Steroid therapy
- Malnutrition
- Sepsis
- Diabetes
- Cancer chemotherapy

**Q27. What is a burn? Enumerate the types of burns and classify burn injuries.**

**Ans.** A burn is coagulative necrosis of living tissue that produces various wounds.

**Causative classification or types of burns**

- Classical burns—due to fire, heat, bomb blasts, etc.
- Chemical burns—due to acidic or basic chemical such as hydrofluoric acid, lye, etc.
- Scalds—hot liquid or steam induced burns
- Electrical burns
- Radiodermatitis
- Cold burns
  - **Frost bite:** Exposure to freezing temperature which lead to formation of ice crystals in tissue.
  - **Chilblain:** Exposure to damp and cold but nonfreezing weather.
  - **Trench foot:** Prolonged exposure to extreme cold weather accompanied by circulatory compromise due to tight outfits.
  - **Immersion foot:** Trench foot occurring in people who spend prolonged time in shipwrecks or waterlogged boats.

**Classification of burns according to depth involved**

<b>First degree</b>	Affect only the superficial layers of epidermis Present with localized redness and edema without any blebs Is painful Heal without scarring Not considered while estimating the magnitude of burns
<b>Mild second degree</b>	Entire epidermis and superficial dermis is destroyed Present with blebs and vesicles Is painful Dermal appendages are preserved and so heals slowly with/ out scarring but on its own
<b>Severe second degree</b>	Entire epidermis and deep dermis is destroyed Present with blebs and vesicles Is painless Dermal appendages are lost and so does not heal on its own and requires skin grafting
<b>Third degree</b>	Complete destruction of epidermis and dermis and deep subcutaneous tissues to variable extent without involvement of underlying soft tissue, bone or joint capsule
<b>Fourth degree</b>	Involve underlying soft tissue, bone or joint capsule

**Classification according to depth**

- **Superficial partial-thickness burns**
  - Burns upto papillary dermis
  - Blistering, hyperemia are present
  - Pinprick sensation is normal
  - The capillary refill is present
  - Heal without residual scarring in 2 weeks.
- **Deep partial-thickness burn**
  - Upto deep reticular dermis
  - Fixed capillary staining is present but capillary refill is not present
  - Sensation lost to mild pain but present to deep pressure or painful stimuli
  - Take longer time to heal without surgery and hypertrophic scarring occurs.
- **Full-thickness burns**
  - The whole of the dermis is destroyed in these burns
  - No capillary staining or return
  - Complete loss of pain sensation
  - Always require surgery to graft and heal.

**Q28. Write a note on assessment of size or percent area of burns.**

**Ans.** Assessment of depth of burns can be done in following ways.

All second and third degree burns are taken into consideration while calculating the burnt area.

1. **Wallace rule of nines** is the commonest method used for all patients greater than 4 years of age and calculates as follows:

Head, face and neck	9%
Anterior trunk (Chest + abdomen)	18%
Posterior trunk (Chest + abdomen)	18%
Each upper extremity	9%
Each lower extremity (Thigh 9% and leg + foot 9%)	18%
External genitalia	1%

**Other methods**

2. **Use a piece of paper** the size of the hand of the patient. That corresponds to 1%.
3. **Lund and Browder charts** are used in children upto 4 years of age.
4. **Berkow formula** is also useful in children.

**Q29. Explain the prehospital care that needs to be administered to a patient with burns.****Q30. Discuss the management of a patient with 40% TBSA burns.****Q31. Discuss the inhalational injuries in a patient with burns.**

**Ans.**

**Prehospital care**

- First step is to ensure personal safety in the field



- Extinguish the fire burning on a person
- Use the **advanced trauma life support protocol** to take care of the patient
  - Airway with cervical spine protection
  - Breathing
  - Circulation
  - Disability and environmental control
  - Exposure and evaluation
  - Rapid secondary survey
- Cool the burn wound with running tap water for a minimum of 10 minutes as this provides analgesia and slows the delayed microvascular damage that can occur after a burn injury
- Elevation of burned limbs.

#### **Management of a patient with 40% TBSA burns**

- Prehospital care as explained above
- ATLS protocol should be repeated in the hospital.

#### **Airway**

- Any patient with change in voice or hoarseness, history of being trapped in smoke filled space, Seinging of hair in nose and stridor should be evaluated critically for **early elective intubation**
- Upper airway burns cause damage to the lining mucosa and this cause edema of these areas and airway compromise
- Also all patients with traditional **indications of intubation** and upper airway burns should be intubated.

#### **Breathing**

- All patients with lower airway burns, breathing difficulties, eschars on chest should be managed for breathing first before proceeding to further management
- Lower airway involvement by steam and fumes of smoke cause edema of lower respiratory tract and impaired exchange of gases
- The fumes contain carbon monoxide which in itself is toxic to body and adds to the insult.
- Also patients can have mechanical obstruction of airway which needs to be taken care of
- Once breathing is established initially, serial clinical examinations, serial blood gas analysis and pulse oximetry readings help determine the status of breathing and ventilation in the patient.

**Circulation:** Taking care of fluid, electrolyte and acid base status.

#### **Indications for fluid therapy**

- **Oral fluids with salt supplement:** Burns less than 10% TBSA in children and less than 15% TBSA in adults.
- **Parenteral fluids:** All other patients.
- **Blood transfusion:** All patients with more than 20% TBSA full thickness burns or more than 40% TBSA partial thickness burns should be considered for blood transfusion as a part of resuscitation.

**Formula for fluids**

1. **Parkland formula** is the most commonly used formula for crystalloids.  
Fluid requirement in ml =  $4 \times \text{TBSA} \times \text{Body weight}$ . The value obtained is divided into 2 parts with half given in first 8 hours and the remaining given in next 16 hours.  
The further fluid requirement after 24 hours is calculated according to the normal patient management formula.

*Other formulas for crystalloids include*

2. Moore formula, Brooke's formula, Evan's formula for adults.
3. **Holiday Segar formula for children**  
Fluid requirement in ml = 100 mL/kg/24 hours for first 10 kg, 50 mL/kg/24 hours for next 10 kg and 20 mL/kg/24 hours for each kg weight thereafter.
4. Galveston formula for children.
5. **Formula for colloids: Muir and Barclay formula**  
 $0.5 \times \text{TBSA} \times \text{Weight} = 1$  portion. This amount given over 4 hours upto 12 hours, over 6 hours upto 24 hours and over 12 hours in the end.

**Management of the burn wound**

After cooling the wound for 10–15 minutes with running water or saline, the following measures are taken:

- Always remember that skin is the best dressing and therefore the aim of the wound management is restoration of skin continuity

**For partial thickness wounds** – Antiseptic cream **impregnated dressings** or **exposure** and limb elevation without any dressing or **biological dressings and skin substitutes** are the best measures and often the only measures required (**Refer the section on wound healing for more on biological dressings and skin substitutes**).

- **Mafenide acetate:** Can penetrate eschar. Cause metabolic acidoses due to carbonic anhydrase inhibition.
- **Silver sulphadiazine**
- **Silver nitrate:** Can cause hyponatremia and methemoglobinemia.

**For full thickness burns**

*Escharotomy*

- Eschar is circumferential full thickness burn that can lead to compression of the blood supply of a limb and compartment syndrome and needs emergent management
- **Indication** – Circumferential deep second degree, third degree and fourth degree burns with impending or established compartment syndrome in chest/abdomen/limbs
- **Signs of compartment syndrome**
  - Limbs: pulselessness, pallor, pain, paresthesia
  - Abdomen: respiratory discomfort, decreased urine output, hypotension
  - Thorax: progressive respiratory distress and falling saturation with hypoventilation and hypotension.
- **Doppler signals and capillary refill time** are also parameters useful to diagnose compartment syndrome in the presence of eschar
- Can be done without anesthesia as the area is insensate

- Done using **tangential excision procedure** commonly as this procedure allows the removal of the burnt eschar till the bed and margins starts oozing.

### Dressing

- **Early excision of the burnt tissue followed by skin grafting** is the best management option and is carried out at 24–72 hours after adequate fluid resuscitation.
- **Dressing of the wound** with above mentioned creams is the next best measure or temporary measure till the patient is prepared for early excision and grafting.

### Other management issues

- Tetanus prophylaxis
- Patient monitoring in ICU setting with charting of temperature, pulse, blood pressure, urine output, intake of fluids and respiratory rate. Frequent sugar levels, arterial blood gas analysis and electrolyte levels are also necessary in critical patients
- Stress ulcer (curling ulcer) prophylaxis for all patients with more than 30–40% TBSA burns
- Nasogastric decompression for all patients with more than 20% TBSA burns as these patients commonly have paralytic ileus
- Nutrition
  - Currerie (calories), Sutherland (calories) and Davies (protein) are some of the formulas available for calculation of the metabolic needs of these patients.
  - For a patient with more than 40% TBSA burns, about 2000 kcal/ body surface area/ day is required with 20% of calorie requirement being given through protein. They also need to be given 500–1000 mL fat emulsion/week.
- Adequate analgesia
- Antibiotic prophylaxis or culture based antibiotics
- Limb elevation, physiotherapy
- Deep vein thrombosis prophylaxis if indicated
- Psychological support.

### Q32. Write a note on pathophysiology of classical thermal burns.

**Ans.** Pathophysiology of burns is divided into local and systemic pathophysiology.

#### Local factors

- Extent of burns (Explained above)
- Depth of burns (Explained above)
- Presence or absence of infection and foreign body
- Vascular changes.

#### Described in the form of three zones in and around the burn area

##### *Zone of coagulation*

- This is the area with irreversibly damaged tissue or the actual burn area.

##### *Zone of stasis*

- The area surrounding the area of coagulation necrosis is this zone which has decreased perfusion and is a zone which can survive with management or undergo coagulative necrosis if not properly managed

- Neutrophil mediated injury and local cytokines and mediators are the key players in this zone
- Treatment directed at the control of local inflammation.

#### *Zone of hyperemia*

- Area surrounding the zone of stasis with vasodilation from inflammation surrounding the burn wound
- It is from here that the healing process begins and is generally not at risk for further necrosis.

#### **Systemic factors**

- Hypoxia, hypercarbia, methemoglobinemia or carboxyhemoglobinemia
- Any form of shock be it hypovolemic, neurogenic, cardiogenic or septic can occur
- Anemia
- Electrolyte imbalances such as hyponatremia, hyperkalemia
- Hypoproteinemia, hyperglycemia or hypoglycemia
- Rhabdomyolysis
- Curling ulcers
- Delirium or depression
- Bilateral adrenal necrosis and hypoadrenal shock can result from vascular thrombosis
- Kidney damage due to low perfusion, rhabdomyolysis, hemoglobinuria and tubular deposits and so on can lead to uremia and renal failure. Therefore monitoring of urine output and maintaining it on higher side is of utmost importance.

### **Q33. Write a note on management of a patient with cleft palate.**

**Discuss the development of lip and palate and classify its developmental clefts.**

**Write a note on cleft lip.**

**Ans.**

#### **Embryology**

##### *Processes*

- Frontonasal process gives rise to median nasal process and lateral nasal process.
- The median nasal process gives rise to two globular processes and a premaxilla.
- The mandibular process gives rise to maxillary process which gives rise to the palatine process.

##### *Fusions*

- Maxillary process + lateral nasal process = cheek  
Defect = facial cleft.
- Median nasal process + maxillary process = lateral part of upper lip.  
Defect = lateral cleft lip.
- Two globular processes = philtrum.  
Defect = central cleft lip.
- Premaxilla + both palatine processes = Hard palate.  
Defect = cleft palate.

- The site of fusion of the three processes forming the palate = incisive foramen.  
The part of palate anterior to incisive foramen = primary palate.  
The part of palate posterior to incisive foramen = secondary palate.

### Types of clefts

- Complex cleft = cleft lip + cleft palate
- Complete cleft = both the median and lateral nasal process are not fused to the maxillary process resulting in cleft lip and a nasal defect
- Compound cleft = cleft associated with alveolar defect
  - The most common type of defect is complex defect
  - The incidence is more in children of affected parents
  - It is more common in males.

### Management

- Do presurgical infant orthopedics such as nasoalveolar moulding. These procedures convert complete defects into incomplete defects and help in better alveolar approximation. If nasoalveolar moulding cannot be performed, do cleft lip adhesion till the child is prepared for surgery
- Cleft lip repair is done at 3–6 months age. The Millard rule of 10 (10 week age, 10 pounds weight and 10 g/dL hemoglobin) is still valid
- Cleft palate repair is done at 9–12 months.

<b>Unilateral cleft lip</b>	<ul style="list-style-type: none"> <li>• <b>Millard</b> rotation – advancement repair. This is the preferred technique</li> <li>• <b>Mirault-Blair, Tennison or LeMuserier Z</b> plasty</li> <li>• After the flap procedures, the lip is sutured in three layers – mucosa to mucosa, muscle to muscle and skin to skin</li> <li>• Add <b>Gingivoplasty</b> if alveoli is involved, <b>Orthodontics</b> and <b>primary nasoplasty</b> if necessary</li> </ul>
<b>Bilateral cleft lip</b>	<ul style="list-style-type: none"> <li>• After presurgical infant orthopedic measures, <b>Mullikan technique</b> of repair of bilateral cleft lip is performed for repair</li> <li>• Add <b>Gingivoplasty</b> if alveoli is involved, <b>Orthodontics</b> and <b>primary nasoplasty</b> if necessary</li> </ul>
<b>Cleft palate</b>	<ul style="list-style-type: none"> <li>• <b>Components of repair</b> include nasal lining, oral mucosal lining, levator palatine muscle and velopharyngeal competence (the closure of oronasal passage during swallowing) and alveolar bone grafting (iliac crest)</li> <li>• For hard palate, <b>Wardill</b> unipedicled mucoperiosteal flap repair or <b>Langenback</b> bipediced flap repair (Both are based on greater palatine vessel) is performed</li> <li>• For soft palate, <b>Straight repair</b> or <b>Furlow's palatoplasty</b> (Z plasty) is done alongwith levator palatine repair called intravelar veloplasty with either of the two procedures</li> <li>• If there is velopharyngeal incompetence after the above repairs of palate, than <b>Wardill pharyngoplasty</b>, <b>Dennis Brown Pharyngoplication</b> or pharyngeal flap technique is used to take care of the defect</li> <li>• Patients with associated maxillary hypoplasia can undergo <b>LeFort I maxillary advancement</b> procedure</li> </ul>

- Postoperatively, the arms and hands of the infant should be restrained to avoid him touching the repair, lip suture line should be splinted if necessary, internal or external nasal fixation to restore normal nasal appearance should be done.
- Local spray of antibiotics as well as systemic administration.
- Spoon and dropper feeding initially and suckling allowed after 2–3 weeks.
- Sutures are removed at 5–6th day.
- Speech training is given as the age advances.

## HEAD AND NECK SURGERY

### Q34. Write a note on anatomy of neck.

#### Ans. Anatomy of neck

It is divided into anterior and posterior triangles by *sternocleidomastoid (SCM)* muscle.

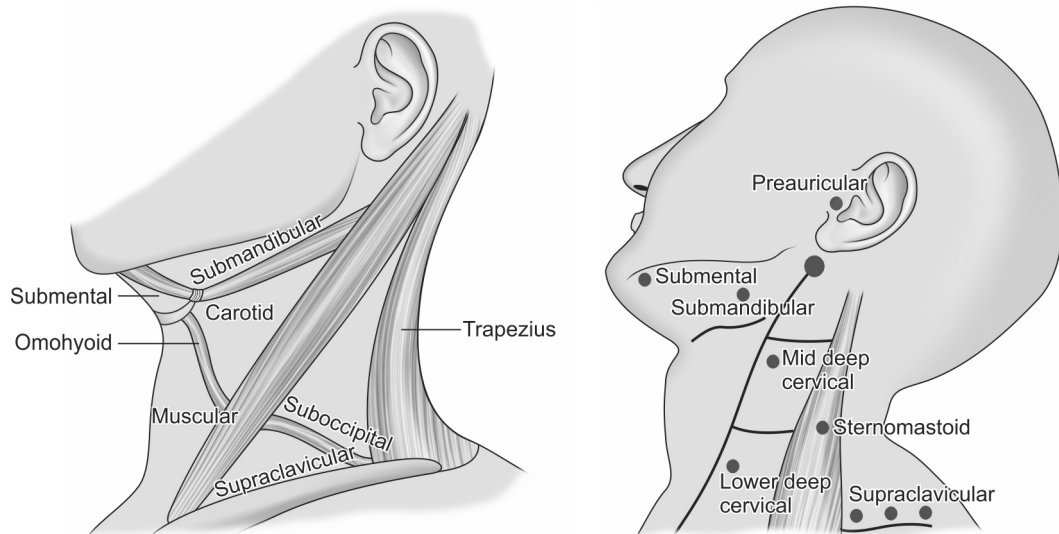


Fig. 1: Anatomy of neck

#### Anterior Triangle

##### Boundaries

- *Lateral:* Anterior border of SCM
- *Anterior:* Anterior midline of neck
- *Superior:* Inferior mandible.

It is divided into four smaller triangles for descriptive purposes.

##### Submandibular triangle

- Between inferior mandible and anterior and posterior bellies of the digastric muscle
- Contains submandibular gland, submandibular duct, and submandibular lymph nodes.

*Submental triangle*

- Between body of hyoid bone and right and left anterior bellies of the digastric muscles
- Apex is mandibular symphysis
- Contains submental lymph nodes.

*Carotid triangle*

- Bounded by anterior belly of omohyoid, posterior belly of digastric, and anterior border of SCM
- Contains carotid sheath, with common carotid artery, internal jugular vein, and vagus nerve, bifurcation of common carotid to internal and external carotid arteries, carotid sinus, carotid body.

*Muscular triangle*

- Bounded by anterior border of SCM, superior belly of omohyoid, midline of neck
- Contains infrahyoid muscles, thyroid, parathyroid.

**Posterior Triangle***Boundaries*

- *Posterior:* Anterior border of trapezius
- *Anterior:* Posterior border of SCM
- *Inferior:* Medial third clavicle
- *Roof:* Investing layer of deep cervical fascia
- *Floor:* Muscles—splenius capitis, levator scapulae, middle scalene, posterior scalene.

*Contains*

- External jugular vein
- Subclavian vein
- Third part of subclavian artery
- Transverse cervical artery (from thyrocervical trunk)
- Suprascapular artery (from thyrocervical trunk)
- Occipital artery (from external carotid)
- Accessory nerve (cranial nerve (CN) XI)
- Ventral rami (roots) of brachial plexus
- Cutaneous branches of cervical plexus
- Suprascapular nerve
- Phrenic nerve.

Posterior triangle is subdivided by inferior belly of omohyoid into,

*Occipital triangle*

- Larger triangle superiorly
- Crossed by accessory nerve.

*Supraclavicular triangle*

- Smaller inferior triangle
- Contains external jugular vein, suprascapular artery, and subclavian artery.

**Facial layers of neck**

**Superficial fascia:** Between dermis and investing layer of deep fascia

**Deep fascia** consists of three layers

- Investing
- Pretracheal
- Prevertebral

Also includes carotid sheath: condensation of deep fascia around carotid vessels.

**Facial spaces***Retropharyngeal space*

- Largest and most significant space in neck
- Potential space between prevertebral layer of deep fascia and buccopharyngeal fascia
- From base of skull to posterior mediastinum
- Permits movement of pharynx, larynx, trachea, and esophagus during swallowing
- Infection originating in pharyngeal area can spread to retropharyngeal space and inferiorly into superior mediastinum.

*Pretracheal space*

- Space between investing fascia and pretracheal fascia
- Limited by attachments of fascia to thyroid cartilages superiorly
- Can spread into thorax anterior to pericardium.

*Space between laminae of prevertebral fascia*

- Critical space
- Extends from base of skull and through thorax

**Lymphatic drainage**

- Level I : Submental and submandibular group
- Level II : Upper jugular group
- Level III : Middle jugular group
- Level IV : Lower jugular group
- Level V : Posterior triangle group
- Level VI : Central group
- Level VII: Upper mediastinal group.

**Q35. What is epulis? How is it different from odontomes? Discuss the types of epulis.**

**Write a note on odontomes.**

**Write a note on epulis.**

**Write a note on adamantinoma.**

**Ans.** (I always remember this topic as a “3 star” topic. 3 types of epulis (4th is false epulis), 3 names of the epulis starting with G, 3 types of odontomes and 3 types of epithelial odontomes and also it has been asked many times in the examinations so 3 star that way also)

- Epulis is a mucoperiosteal swelling located on the gums (**Mucus membrane—Carcinomatous type, Bone—myeloid type and Periosteum—Fibrous type**)



- It is different from odontome because odontome arises from the tooth germ and can be **epithelial odontome** such as dental cyst, dentigerous cyst or adamantinoma or **connective tissue odontome** or **composite odontome** comprising of both the epithelial and connective tissue odontomes
- **Types of epulis and their salient features are as follows:**

<b>Fibrous epulis</b>	<ul style="list-style-type: none"> <li>• Most common type</li> <li>• Arises from periosteum</li> <li>• Most common site is incisor or premolar tooth</li> <li>• Slow growing, firm, polyp like lesion that is nontender and prone to recurrence</li> <li>• Can turn malignant – fibrosarcoma</li> <li>• <b>Treatment</b> is resection with tooth and adjacent bone excision</li> </ul>
<b>Myeloid epulis</b>	<ul style="list-style-type: none"> <li>• Arises from bone and contains giant cells and has the soap bubble appearance similar to osteoclastoma</li> <li>• It never turns malignant</li> <li>• It is flat tumor with increased vascularity so can bleed profusely spontaneously and during surgery</li> <li>• <b>Treatment</b> is curettage of small lesions whereas excision with bone resection for bigger lesions</li> </ul>
<b>Granulomatous epulis</b> (Gingivitis gravidarum as it is sometimes seen during pregnancy) (Remember: 3G epulis)	<ul style="list-style-type: none"> <li>• It is composed of granulation tissue at site of dental caries or site persistently irritated due to tooth speck or denture</li> <li>• Therefore it does not actually arise from mucoperiosteum and so is called false epulis</li> <li>• As it is granulation tissue, it looks same as it and also bleeds to touch</li> <li>• Can be associated with enlarged lymph nodes due to the causative factors</li> <li>• <b>Treatment</b> is cauterisation of the granulation tissue and excision of the affected tooth and correction of cause</li> </ul>
<b>Carcinomatous epulis</b>	<ul style="list-style-type: none"> <li>• This arises from mucus membrane and is an alveolar epithelioma</li> <li>• It is locally invasive, painful and can get infected or ulcerated</li> <li>• Lymphadenopathy is associated due to malignancy or infection</li> <li>• <b>Treatment</b> is resection with adequate margin with mandible/ maxilla resection as deemed necessary</li> </ul>

**The odontomes include the lesions mentioned above and are as follows:**

Dental cyst	Dentigerous cyst
<ul style="list-style-type: none"> <li>• It is also known as radicular cyst or periodontal cyst</li> <li>• Most common type</li> <li>• Arises in relation to chronically infected normally erupted tooth</li> <li>• Always occurs in upper jaw</li> <li>• Occurs in middle age</li> <li>• Can be painless or painful</li> <li>• Radiolucent area is seen at root of tooth on X-ray</li> <li>• Treatment is total excision of cyst as well as tooth alongwith complete curettage of the epithelial lining.</li> </ul>	<ul style="list-style-type: none"> <li>• It is also known as follicular odontome or follicular cyst</li> <li>• Second most common</li> <li>• Arises in relation to nonerupted permanent tooth.</li> <li>• Can occur in relation to upper or lower third molar but lower jaw is more commonly affected.</li> <li>• Occurs in young age (2nd or 3rd decade)</li> <li>• Always painless</li> <li>• Radiolucent area is seen at crown of tooth on X-ray</li> <li>• Treatment is excision or marsupialization. Epithelial curetting is not necessary</li> </ul>

Dental cyst	Dentigerous cyst
<b>Common points</b>	
<ul style="list-style-type: none"> <li>• Both are benign lesions</li> <li>• Both these lesions cause thinning of the outer bone cortex and lead to egg shell cracking</li> <li>• Both have lining of squamous epithelium and contain liquid or semisolid material</li> <li>• Maintenance of dental hygiene and prevention of infection are important in management of both the lesions</li> </ul>	

### Adamantinoma

- It is also an epithelial odontoma, more specifically an epithelial neoplasm of germ of teeth—the ameloblasts (Enamel forming cells)
- It is also known as ameloblastoma or Eve's disease or multicystic disease of jaw
- It is seen in childhood or early second decade and is more common in males
- It more commonly affects outer table of the lower jaw in molar area
- It is painless, slowly growing mass with egg shell cracking due to thinning of outer table
- **Pathologically**, it is composed of columnar cell lining with inner lining of different cells and cystic degeneration
- **X-ray** shows honey comb appearance with thinning of outer cortex. The bony trabeculae are normal
- **Differentiated** from the other jaw tumors based on these points
  - Osteoclastoma has soap bubble appearance and it affects both tables of bone. Also, trabeculae are poorly defined and pathology shows giant cells
  - Giant cell granuloma is more common in females and affects both tables of bones. Also, trabeculae are absent in the involved area
- **Treatment** is resection with a good margin with/without mandibular resection
- Recurrence is common if not excised completely.

### Q36. Enumerate the premalignant lesions of the oral cavity. Discuss leukoplakia in brief.

**Ans. The premalignant lesions for oral cavity malignancy are as follows:**

- Erythroplakia is associated with maximum risk
- Speckled leukoplakia or erythroleukoplakia
- Leukoplakia
- Oral candidiasis
- Oral submucosal fibrosis
- Plummer Vinson syndrome
- Syphilitic glossitis
- Lichen planus
- Dyskeratosis congenita
- Lupus.

### Leukoplakia

- White lesion of oral mucosa that is not any other diagnoses
- Basically, it is a diagnoses of exclusion
- It is a premalignant condition but the risk is several times less than that of erythroplakia.

**Risk factors**

- Most often the cause is not known, i.e. idiopathic.

Factors commonly implicated include

- Oral tobacco consumption in any form
- Injury with Jagged tooth or ill-fitting denture
- Alcohol
- Oral syphilis
- Oral hairy leukoplakia is caused by Epstein Barr virus.

**Pathology**

- Leukoplakia can be homogenous or inhomogenous
- Homogenous leukoplakia is slightly elevated, plateau like lesion with regular margins
- Inhomogenous leukoplakia has irregular surface and margins and has more chances of malignant transformation. Verrucous leukoplakia is a variant of inhomogenous leukoplakia and has more chances of malignancy
- Erythroleukoplakia or speckled leukoplakia is mixed variant of leukoplakia and erythroplakia and is also premalignant
- It has areas of hyperkeratosis or perakeratosis.

**Diagnoses**

- Biopsy.

**Treatment**

- Eradication of risk factor
- Excision of the lesion if it does not regress after risk factor elimination.

**Q37. Write a note on Cystic hygroma.**

**Ans.**

- It is a congenital malformation of lymphatic system
- It is remnant of sequestered primitive lymph sac and so is a hamartoma—a type of lymphangioma
- Incidence = 1 in 12000 live births.

**Clinical features**

- It is seen in infants prenatally, at birth or early after birth
- Most common site—Nape of neck (Posterior triangle)
- Other sites—Floor of mouth, axilla, mediastinum, retroperitoneum and inguinal region.
- On examination, it is a cystic loculated swelling with ill-defined margins, filled with clear lymph fluid, can be large, is soft in consistency, fluctuant and brilliantly transilluminant with increase in size on coughing, straining or crying
- Always examine the other possible sites to see if they also have the cystic hygroma.

**Complications**

- Infection
- Hemorrhage
- Airway compromise
- Recurrence after treatment—the rate is 20% even after complete resection.

**Management**

- CECT neck and chest is enough to diagnose.
- Prenatal diagnoses with ultrasound before 30 weeks is associated with increased risk of fetal hydrops, associated chromosomal anomalies and polyhydramnios.
- A large cystic hygroma at the base of tongue/ floor of mouth can cause severe airway compromise at birth. When such a case is anticipated, **Ex-utero intrapartum therapy (EXIT)** can be used. Here, 20–30 minutes of placental circulation can be obtained by deep uterine relaxation by general anesthesia and uterine perfusion with warm saline. Then orotracheal intubation or emergency tracheostomy can be done while the infant is still attached to placenta. This is a domain of fetal surgery.
- **Treatment after birth has following options:**
  - Complete surgical excision is the best management.
  - Injection sclerotherapy with Bleomycin.
  - Combined resection and sclerotherapy.

**Q38. Write a note on hemangioma.****Ans.**

- It is a congenital malformation of blood vessels.
- Hemangioma is a hamartoma.
- The **types of hamangioma** are as follows.

<b>Capillary hemangioma</b>	<ul style="list-style-type: none"> <li>• <b>Spider nevus</b> is a sign of liver failure and seen on shoulder or anterior chest</li> <li>• <b>Salmon pink patch</b> on forehead since birth— disappears by age 1 year</li> <li>• <b>Portwine stain</b> on face, never spontaneously regresses</li> <li>• It is associated with syndromes such as               <ul style="list-style-type: none"> <li>– Klippel Treanaunay syndrome (has malformations from all arteries, veins and lymphatics),</li> <li>– Osler Weber Rendau disease (Multivisceral hemangioma)</li> <li>– Sturge Weber syndrome (Leptomeningeal and ocular involvement)</li> </ul> </li> <li>• <b>Strawberry angioma</b> follows the characteristic phases of hemangioma as described below</li> </ul>
<b>Venous hemangioma</b>	Cavernous hemangioma
<b>Arterial hemangioma</b>	Plexiform hemangioma

- It has female preponderance.
- On examination, it is diffuse, reddish or brown slightly elevated or flat lesion with irregular margins and surface.
- **Phases**
  - Proliferating phase—up to 1 year
  - Involuting phase—up to 5 years
  - Involution phase—50% by 5 years and 70% by 7 years.
- Kaposiform hemangioma can cause platelet trapping and bleeding disorder which is called **Kassabach-Merritt syndrome**
- Children with more than 3 cutaneous hemangiomas should have abdominal ultrasound to rule out multivisceral involvement

- **Problematic or endangering hemangiomas** are periocular hemangiomas threatening vision, airway hemangiomas and facial disfiguring lesions
- **Complications**
  - Hemorrhage
  - Infection
  - Calcification
  - Malignant transformation.
- **Management**
  - **First line**
    - Systemic corticosteroids
    - Has 80–90% success rate.
  - **Second line** treatment – Systemic interferon and vincristine
  - **Third line** treatment – Surgery to clear the residual fibrofatty deposits and for the treatment of secondary deformities
  - Therapeutic embolization, ligation of the feeding vessel and excision and skin grafting are other options.

**Q39. Write a note on branchial cyst and fistula.**

**Ans.**

**Embryology**

- In all there are 6 branchial components from each of the three germ layers.
  - Branchial pouch – Endoderm
  - Branchial arch – Mesoderm
  - Branchial cleft – Ectoderm.
- Normally, the second arch grows over 3rd, 4th and 5th arch to fuse with 6th arch and finally all this disappears except for the structures that they form. If these fusions persist, they form cystic collections of the ectodermal secretions called branchial cyst
- If the fusion fails in the first instant, it results in branchial fistula. It can also result from infection and rupture of branchial cyst.

**Branchial cyst**

- The pathophysiology is as described above
- As the inner surface is ectoderm, it is lined by squamous epithelium and contents are ectodermal secretions and cholesterol crystals
- It makes its appearance in the second or third decade
- **On examination**, second arch branchial cyst is located at middle of the anterior border of sternomastoid
- It can be large, nontender, cystic and fluctuant swelling with a smooth surface and well defined margins with limited mobility. Transillumination is negative and it grows very slowly.
- **Complications**
  - Infection
  - Rupture and fistula formation
- **Treatment** is surgical excision.

**Branchial fistula**

- Pathophysiology is as described above.
- Second arch fistula is the most common.
- **Route of fistula as per arch**

<b>1st branchial remnant</b>	External opening is in parotid region.	The other opening is in the external auditory canal	This represents the anatomy of the 1st branchial components (Ear, mandible, etc.)
<b>2nd branchial remnant</b>	External opening is at middle of the anterior border of sternomastoid	The other opening is in the tonsillar fossa	The route passes through between the internal carotid artery posteriorly and external carotid artery anteriorly at the carotid bifurcation. It also passes between the stylomandibular ligament and stylopharyngeus muscle to connect the two openings
<b>3rd branchial remnant</b>	This also opens at the anterior border of sternomastoid at lower aspect	The other opening is at the pyriform sinus	Its tract passes posterior to the carotid bifurcation to connect the two openings

**Diagnoses** – Fistulogram from the external opening will demonstrate the entire tract and help plan the surgery.

**Treatment** is complete excision of the fistula tract to prevent the rare occurrence of branchogenic carcinoma using **step ladder incisions**. That is from the external opening, parallel horizontal incisions are given at increasing level till the entire tract is completely accessible and excised.

**Q40. Write a note on ranula.**

**Ans.**

- Ranula is derived from rana—frog
- **Pathophysiologically** it is a mucus retention cyst of secretions of minor salivary glands in the floor of the mouth
- **Clinically**, it is seen in second or third decade
- It is a painful or painless, soft, cystic swelling in the floor of mouth that resembles frog belly and so the name
- It is transparent bluish swelling that is fluctuant and transilluminant
- It can present as neck swelling in the submandibular region alongwith oral swelling and the variety is called plunging ranula
- **The diagnoses** is by examination and bidigital palpation should not be forgotten to avoid missing the plunging variety
- **Treatment** is complete excision
- Incomplete excision can result in recurrence
- Simple oral ranula is approached from within the oral cavity whereas the plunging variety is operated from neck
- Marsupialization to the oral mucosa at the floor of the mouth is other option if the lesion cannot be excised completely.

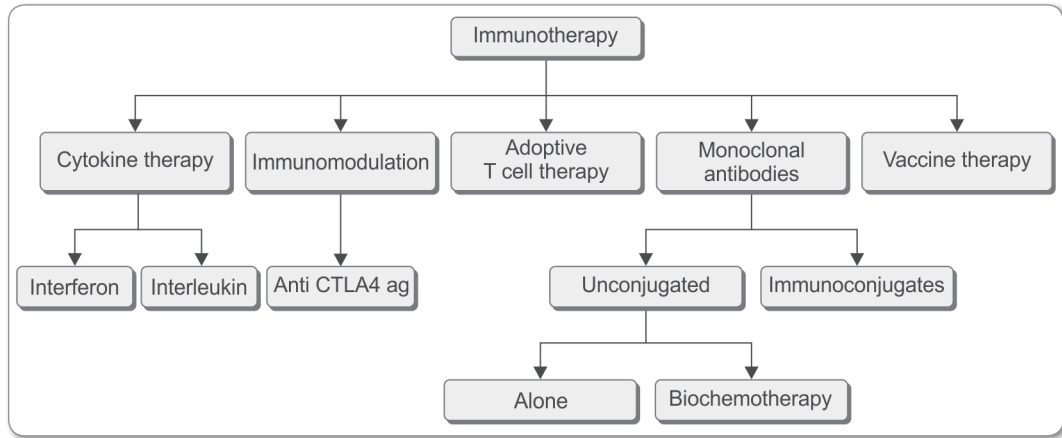
## ONCOSURGERY BASICS

**Q41. Write a note on immunotherapy in cancer.**

**What is immunotherapy? Discuss its types.**

**Write a note on role of monoclonal antibodies in cancer patients.**

**Ans.** Groups of immunotherapy in cancer is as shown in the chart below:



### Interferon

#### Types

Type 1	Alfa, beta
Type 2	Gamma
Type 3	Lambda

#### Effects

<b>On innate immunity</b>	Activation of natural killer and lymphokine activated killer cells
<b>On adoptive immunity</b>	<ul style="list-style-type: none"> <li>• <b>MHC</b> presentation is increased</li> <li>• <b>Th1</b> production increased</li> <li>• Antibody production enhanced</li> </ul>
<b>On tumor cells</b>	<ul style="list-style-type: none"> <li>• Cytostatic / toxic effect on tumor cells</li> <li>• Cause adhesion molecule expression</li> </ul>
<b>Non immune effects</b>	Antiangiogenic effect (anti VEGF) effect

#### Uses

- Metastatic melanoma
- Metastatic renal cell carcinoma
- Hairy cell leukemia, non-Hodgkin's lymphoma
- Kaposi sarcoma
- Infantile hemangioma, epithelioid hemangioendothelioma, giant hemangioma
- Chronic myeloid leukemia

- Hepatitis C
- Giant cell tumor of mandible.

### Toxicities

- Constitutional
- Supraventricular tachycardias
- Atrial fibrillation
- Retinopathy
- Autoimmune diseases such as thyroiditis, rheumatoid arthritis, Raynaud's disease
- Depression or hypomania.

### Interleukin 2

- Used in metastatic melanoma, metastatic renal cell carcinoma
- Administered with or without lymphokine activated killer cells.

### Vaccines

These can be

- Whole cell vaccine
- GM-CSF modified tumor vaccines
- Signalling peptide based vaccines
- Dendritic cell based vaccines (prostate monocyte loaded with prostatic acid phosphate-GM-CSF fusion protein)
- DNA based vaccine.

None have been successful in trial so far.

### T cell adoptive therapy

T cell adoptive therapy is administered in the following way

- Firstly, tumor infiltrating leukocytes (TIL) from metastatic sites (mainly melanoma) stimulated by interleukin-2 are isolated.
- This helps in collecting T cells
- After inducing near total T cell depletion in recipient, inject  $5 \times 10^{10}$  cultured TILs with systemic interleukin-2 with or without total body irradiation (200–2000 cGy).
- This changes the recipient lymphocyte count and increases the donated pool and therefore is called adoptive T cell immunity.

### Monoclonal antibodies

*Types*

Murine	Fully mouse in hybridoma
Chimeric	Fc is human
Humanized	Human with murine variable region
Fully human	Fully human immunoglobulin
Genetically engineered	Genetically engineered (fragmented m ABs)
Unconjugated	
Immunoconjugates: targeted radioimmunotherapy	



In radio immunotherapy, monoclonal antibodies are combined to radionuclides  
Beta emitters use in radio immunotherapy are of two types:

High energy (1 cm penetration)	Yttrium-90, rhenesium-188
Medium energy (1 mm penetration)	Iodine-131, lutetium-177

FDA approved radio immunotherapy

Yttrium-90	Ibritumomab
Iodine-131	Tositumomab

Used in NHL wherein addition of these agents have been shown to increase overall response, increase complete response rates, and durability of response.

### Naming of monoclonal antibodies

They have a 4 syllable name

1	2	3	4
Unique prefix	Indication	o/xi/u human/chimeric/murine	Mab means monoclonal antibody
Eg: Ri	Tu	Xi	Mab

- When only monoclonal antibody therapy is used, it is called unconjugated therapy
- When administered together with chemotherapy, it is called biochemotherapy
- **Examples of biochemotherapy** include trastuzumab in breast cancer with CAF
- Cetuximab, bavacizumab with FOLFOX in colorectal cancer, rituximab with CHOP in non-Hodgkin's cancer.

### Mechanism of action of monoclonal antibodies

- Direct tumor cell surface receptor agonist leading to apoptosis or antagonist inhibiting the signalling for proliferation and inducing apoptosis
- If conjugated, help in delivering drug, radio-isotope or toxin and mediate tumor cell death
- Induction of complement activation, phagocytosis or antibody dependent cytotoxicity
- Vascular or stromal ablation in the mesenchyme of tumor.

## Q42. Enumerate the groups and mechanism of action of the Immunosuppressant drugs.

Ans. Groups and mechanism of action

Group	Drugs	Mechanism of action
Corticosteroids	Methyl prednisolone	Block NF-K beta by enhancing IK beta and block transcription of II1 and TNF alfa.
Calcineurin inhibitors	<ul style="list-style-type: none"> <li>• Cyclosporine (bind cyclophilin)</li> <li>• Tacrolimus (bind FKBP 12, FK-506)</li> </ul>	Both inhibit calcineurin and therefore inhibit IL 2 mediated T cell activation.
Antimetabolites	<ul style="list-style-type: none"> <li>• 6 mercaptopurine (6-MP)</li> <li>• Azathioprine (AZA)</li> <li>• Mycophenolate mofetil (MMF)</li> </ul>	6-MP and AZA inhibit DNA synthesis. MMF is a IMP dehydrogenase inhibitor and inhibits both DNA and RNA synthesis.

Contd...

Contd...

Group	Drugs	Mechanism of action
Lymphocyte depletion preparation	<ul style="list-style-type: none"> <li>• Anti-lymphocyte globulin</li> <li>• Muromonab CD 3 (OKT3)</li> <li>• (anti CD25) include basiliximab and daclizumab</li> </ul>	OKT3 is the first monoclonal antibody approved. It rapidly decreases the number of circulatory T lymphocytes. Anti CD25 molecules are IL2 receptor blocker. Therefore useful in induction.
Costimulation blockade (CD 28)	Belatacept	Bind CD 80/CD 86 and cause costimulation blockade
Immunoglobulin agents	<ul style="list-style-type: none"> <li>• Rituximab</li> <li>• Alemtuzumab</li> <li>• IVIg</li> </ul>	<ul style="list-style-type: none"> <li>• Anti CD20</li> <li>• Anti CD52 cause up to 3 months depletion of T and B cells</li> <li>• Desensitization</li> <li>• Decrease panel reactive antibody test score and decrease incidence of positive crossmatch in transplants</li> </ul>
Newer immuno-suppressants	<ul style="list-style-type: none"> <li>• Fingolimid</li> <li>• Deoxyspergualin</li> <li>• Rapamycin (sirolimus and everolimus)</li> <li>• Belatacept</li> </ul>	

**Newer immunosuppressants**

- **Rapamycin analogues**
  - Obtained from streptomyces hygroscopicus
  - Blocks FKBP and inhibit mTOR. This FKBP-rapamycin associated protein inhibits p70s6 kinase and prevent G1- S phase cell cycle progression
  - Include sirolimus and everolimus
  - Uses in induction immunosuppression, antiproliferative effect utilized for treatment of B cell lymphoma
  - Also used in drug eluting stents and to prevent PTLT
  - Side effects include oral ulcers, hyperlipidemia and hypertriglyceridemia.
- **Belatacept**
  - Block CD 80 and CD 86 and therefore causes costimulation blockade (CD 28)
  - It is a second generation CTLA4 Ig molecule. First generation is called abatacept
  - Both decrease t cell activation.
- **Fingolimid**
  - It is a sphingosine analogue
  - Binds to S-1-P receptor 1 and gets internalized and causes decrease degrees of lymphocytes from secondary lymphoid organs thus, essentially traps lymphocytes in these organs
  - However, not successful in trials so far
  - Still used in multiple sclerosis
  - Side effects—macular edema, nephrotoxicity, bradycardia.
- **Deoxyspergualin**
  - Blocks antigen presenting cells and decrease IL 1 and TNF production
  - Disappointing results so far.

**Side effect profile**

Steroids	Impaired wound healing, diabetes, weight gain, osteoporosis, adrenal suppression
Cyclosporin	Renal toxicity is transient, dose dependent, reversible and is due to vasoconstriction of renal artery Headache, hypertension, hyperuricemia, hyperlipidemia, hirsutism, hepatotoxicity, gum hypertrophy, tremors, seizures
Tacrolimus	Similar to cyclosporine but high incidence of neurotoxicity and diabetes
Sirolimus	Thrombocytopenia, increase toxicity of calcineurin inhibitors
Muromonab CD3	Cytokine release syndrome
MMF	Neutropenia, mild anemia
AZA	Bone marrow suppression
Daclizumab, basiliximab	Hypersensitivity reaction
Rituximab	Hypersensitivity reaction

**Q43. Write a note on cancer prevention.**

**What is screening for cancer? Enumerate the basis for screening.**

**Ans. Primary prevention:** Prevention of initial cancer in healthy individuals.

**Secondary prevention:** Prevention of cancer in patients with premalignant conditions.

**Tertiary prevention:** Prevention of cancer in patients who have been cured of cancer.

Modality of cancer prevention is cancer screening followed by preventive strategy in susceptible population (cancer screening is discussed below).

**Preventive strategy includes the following**

Chemoprevention	Surgical prevention
<b>Tamoxifen/raloxifen</b> in breast cancer	<b>Bilateral mastectomy</b> (BRCA 1/2)
<b>Celecoxib</b> in familial adenomatous polyposis	<b>Orchidectomy</b> (undescended testis)
<b>13 cis retinoic acid</b> in patients with leukoplakia	<b>Thyroidectomy</b> (RET mutation)
	<b>Total colectomy</b> (FAP, Long standing ulcerative colitis or Crohn's disease)
	<b>Total gastrectomy</b> (CDH1 mutation)

**Cancer screening**

Early detection is the key to success in cancer therapy.

Screening for common cancers using relatively noninvasive tests is expected to **lead to**

- Early diagnosis
- Allow more conservative surgical therapies with
- Decreased morbidity, and potentially
- Improve surgical cure rates and
- Improved overall survival rates.

**Key factors that influence screening guidelines are**

- The prevalence of the cancer in the population

- The risk associated with the screening measure, and
- Whether early diagnosis actually affects outcome.

#### The risks involved with the screening

- More invasive screening measures such as colonoscopy
- The consequences of a false-positive screening test  
For example, false positive and false negative rate of screening mammogram is 10%.  
Also,  
Among women for whom biopsy is recommended, 25–40% will have a breast cancer.  
A false-positive screen is likely to induce
  - Significant emotional distress in patients,
  - Leads to unnecessary biopsies, and
  - Has cost implications for the health care system.
- The consequences of a false-negative screening test
- Screening guidelines are developed for the general baseline-risk population. These guidelines need to be modified for patients who are at high risk.

The **test used for screening** should be valid, accurate, sensitive and specific with low false positive and false negative rate, reliable and with no or minimum interobserver variability.

The **disease selected for screening** should have a definite lag period, test which can diagnose it at an earlier stage, curative therapy to limit progression of the disease and should be a common health problem in that community.

American society recommends following screening measures for important cancers in patients with average risk.

Cancer	Guidelines
<b>Breast</b>	Self examination since age 20 yrs 3 yearly clinical examination from age 20–40 Yearly clinical exam from age 40 years onwards Yearly mammogram starting at age 40
<b>Colon and rectum</b>	<b>Discussed in GI oncology section</b>
<b>Prostate</b>	PSA + DRE yearly since age of 50 years.
<b>Skin</b>	To evaluate each skin lesion for Assymetry/ Boder irregularity/ Color changes/ Diameter > 6 mm ( <b>ABCD</b> ) and consult the specialist if any of them is present
<b>Cervix</b>	Yearly (Regular PAP test) or 2 yearly (Liquid based PAP) PAP smear test starting after 3 years of intercourse or after age 21 years till age 30 and 3 yearly till age 70 if all are normal till age 30 yrs. If 3 normal at age 70, patient can stop testing

#### Q44. Write a note on tumor markers.

**What are tumor markers? Enumerate them and mention their clinical significance.**

**Ans.** Tumor markers are biomarkers found in blood, urine or body tissues that indicate the clinical, biochemical or molecular alterations to aid in the detection of presence of neoplasia.

**Types**

Protein markers	DNA specific markers	RNA specific markers	Epigenetic changes
CEA AFP PSA CA 19-9 CA 125 CA 27-29 CA 15-3 HCG, etc.	RET APC BCR-ABL EGFR1 and 2 KRAS p53	Oncotype Dx Mammaprint for breast cancer	Promoter methylation mutations

**Uses**

- Screening of cancer (PSA in prostate cancer) and early detection
- Diagnoses and staging of cancer (AFP, LDH, HCG in testicular cancer)
- Prognosis of cancer (LDH in malignant melanoma, Ca 19-9 in pancreatic cancer)
- To know whether a treatment will work or not ( Her2neu in breast cancer to see whether trastuzumab will work or not, K-RAS in colon cancer to see whether targeted therapy will work or not)
- To detect recurrence (CEA in colon cancer)
- To monitor response to treatment (CEA in colon cancer, PSA in prostate cancer).

**Markers**

Blood	Tumor tissue
<b>Thyroid</b> —calcitonin, Thyroglobulin <b>Breast</b> —Ca 15-3, Ca 27-29 <b>Lung</b> —SMRP (soluble mesothelin related peptide) <b>Pancreas</b> —Ca 19-9 <b>Liver</b> —AFP <b>Colon</b> —CEA <b>Ovary</b> —Ca 125, inhibin <b>Testis</b> —AFP, HCG, LDH <b>Urinary bladder</b> —BTA, NMP 22 <b>Prostate</b> —PSA, PAP <b>Melanoma</b> —S 100 <b>Neuroendocrine markers</b> <ul style="list-style-type: none"> <li>• Neuroblastoma, small cell lung cancer—neuron specific enolase</li> <li>• Pheochromocytoma—catecholamines, VMA, Metanephrines</li> <li>• Carcinoid tumors—Chromogranin A, Synaptophysin</li> </ul> <b>CML</b> – BCR-ABL <b>Multiple myeloma, CLL, Waldenstrom disease</b> —Immunoglobulin	<b>EGFR 1 and 2</b> in head and neck cancer, colon cancer <b>K-RAS</b> — <ul style="list-style-type: none"> <li>• No response to cetuximab and panitumumab in colon cancer</li> <li>• No response to gefitinib and erlotinib in lung cancer</li> </ul> <b>ER and PR</b> in breast cancer <b>BRAF</b> —response to vemurafenib in melanoma, thyroid <b>ALK (anaplastic lymphoma kinase)</b> —predict response of crizotinib in lung cancer

**Q45. Write a note on oncogenes.****What are oncogenes? Enumerate them.**

**Ans.** Proto-**oncogene**: A normal gene which, when altered by mutation, becomes an **oncogene** that can contribute to cancer.

An **oncogene** is a gene that has the potential to cause cancer. In tumor cells, they are often mutated or expressed at high levels.

Oncogenes are gain of function mutations and are dominant to the cell. That is, a single copy is enough to cause mutation.

Oncogenes are classified as follows:

<b>Growth factors</b>	HGF		Thyroid cancer
	SIS (PDGF beta chain) TGF alfa, HST 1, INT 2		
<b>Signal transduction</b>	BRAF		Melanoma
	Beta catenin		HCC
	RAS	K RAS	Colon, pancreas
		H RAS	Thyroid, bladder, kidney
		N RAS	Melanoma
<b>Surface receptor</b>	ABL		Chronic myeloid leukemia
	Her 2 neu		Breast cancer
	cKit		GIST
	RET		MEN 2A and 2B
<b>Transcription activator</b>	C – MYC		Burkitt lymphoma
	N – MYC		Neuroblastoma, small cell lung cancer
	L – MYC		Small cell lung cancer
<b>Cell cycle regulation</b>	Cyclin D/E		Breast cancer
	CDK 4		Melanoma

#### Q46. Write a note on tumor suppressor genes.

**What are tumor suppressor genes? Enumerate them.**

**Ans.** Tumor suppressor gene is a protective gene that normally limits the growth of tumors. These can be **caretaker genes** (Nuclear genes such as p53, Rb, WT1, BRCA 1 and 2, p16/INK4A) or **promoter genes** – all others.

These are **recessive genes** to the cell. That is, they require loss or inherited or somatic mutation of both alleles by either Knudson 2 hit hypothesis or loss of heterozygosity.

**Types of tumor suppressor genes are as follows**

- **Cell surface proteins:** TGF beta receptors, E-Cadherin
- **On the inside of plasma membrane:** NF1 (Ch. 17)- NF1 normally converts GTP bound RAS (active) to GDP bound RAS (inactive). There is increased cellular proliferation when RAS is in active form.
- **Cytoskeleton:** NF 2 (Ch.22)
- **Cytosol**
  - APC—normally inactivates beta-catenin which when active increases cellular proliferation
  - PTEN – P13 kinase signal transduction
  - SMAD 2 and 4 (effector molecules in TGF beta pathway).

**Important gatekeeper gene pathways – regulation on cell cycle.**

- **p53** when active acts on p21 and p21 and **p16INK4A** inhibit Cyclin D-CDK4 complex (G1 phase of cell cycle)- this keeps **Retinoblastoma gene** active (hypophosphorylated is active) which inactivates E2F and therefore Cyclin E- CDK2 transcription (S phase of cell cycle) thus inhibiting proliferation of cell
- **BRCA 1 and 2** increase p21 production and act by above pathway as cell cycle inhibitors. It also causes homologous recombinant DNA repair by co-localizing with rad 51 and therefore decreases DNA damage.

Carcinogenesis is thus a multistep process – a combination of both tumor suppressor genes and oncogenes mutation is required.

Cell with tumor suppressor gene mutation is a cell with MUTATOR phenotype, i.e. prone to malignancy and a cell with oncogene mutation is MUTATED phenotype, i.e. it will proliferate to malignancy.

**Q47. Write a note on sacrococcygeal teratoma.**

**Ans.**

- It is a teratoma derived from a primitive streak derivative and arises at the base of coccyx and sacrum
- It is the most common germ cell tumor diagnosed in infants and children younger than 4 years
- It is more common in girls.

**Presentation**

- They can be diagnosed on **prenatal** ultrasound and can be externally growing towards skin or internally growing towards the pelvic organs  
It can lead to fetal hydrops and demise, preterm labor, polyhydramnios. Also, a cesarean section might be necessary for the fetal delivery
- **Neonatal** growths present as sacrococcygeal exophytic teratomas
- **Infants and children** presenting late usually have the internal variety palpable per abdomen or imaging showing displacement of bladder or rectum due to a mass arising from sacrococcygeal region
- The presentation in **older age group** is rare.

**Classification** is given by Altman and is as follows

- Entirely exophytic
- Nearly entirely exophytic
- Mostly to the inside
- Entirely inside. Also called retrorectal teratoma or presacral teratoma
  - The triad of malformed sacrum, anorectal malformation and presacral teratoma is called **Currarino triad**. It is an autosomal dominant condition.

**Malignancy risk**

- Masses presenting at birth or within 2 months of birth are more likely to be benign whereas the masses after this age are more likely to be malignant.

**Diagnoses**

- Symptoms can be a presacral mass, constipation, urinary difficulties, bowel obstruction, hydronephrosis and so on
- A solid or cystic mass on prenatal ultrasound and growing internally or externally
- MRI is useful for difficult cases to diagnose it.

**Treatment**

- Complete surgical removal through a perineal or abdominal approach
- The procedure includes resection of the involved parts of coccyx and sacrum and reattachments of the muscles that are attached to these bony segments in the usual cases. The rare cases in older age group should be resected without the bony resections if possible.



## FURTHER READING

1. Bailey and Love's Short practice of surgery 26th edition.
2. Blumgart's Surgery of the liver, biliary tract and pancreas.
3. Campbell Urology 10th edition.
4. Grainger and Allison's Diagnostic radiology 5th edition.
5. Haaga's CT and MRI of the whole body 5th edition.
6. Harrison's Practice of internal medicine.
7. Paul Marino's The ICU book.
8. Sabiston textbook of surgery 19th edition.
9. Schwartz's principles of surgery 10th edition.
10. Shackelford's Surgery of the alimentary tract 7th edition.
11. Sutton's Textbook of radiology and imaging 7th edition.

*For any further Queries/ Suggestions/Comments/ Guidance in this fascinating field of surgery, Feel free to contact us on*

- [1988ronakpatel@gmail.com](mailto:1988ronakpatel@gmail.com)
- [dssh1sh@yahoo.com](mailto:dssh1sh@yahoo.com)
- [www.facebook.com/Gunjan Desai](https://www.facebook.com/GunjanDesai)
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